

T H E S I S

on

"OBSERVATIONS UPON APPENDICITIS

BASED ON AN ANALYSIS OF

SIX HUNDRED CASES"

Submitted for the degree of Doctor of Medicine

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INTRODUCTION

Notwithstanding the fact that appendicitis is one of the most common diseases met with in everyday practice, the medical attendant has still to face difficult problems regarding its accurate diagnosis, prognosis, and scientific treatment. That "Appendicitis" is not a disease devoid of surgical interest and importance is shown by the diversity of opinion which still exists among surgeons regarding the appropriate line of treatment to be adopted in the various stages of the disease. And that the condition is not one lacking in economic importance is shown by the fact that, in England and Wales, over 2,500 persons die yearly from appendicitis and its complications. In other words, the loss of life every year from appendicitis exceeds that which would result from the occurrence of two Titanic disasters !

A plea, therefore, for a consideration and investigation of the diseases of the vermiform appendix, and their treatment, could be amply justified on the following grounds :-

- (1) Frequency of the disease Appendicitis is one of the commonest ailments which besets the human frame.
- (2) Mortality The mortality from appendicitis is still much too high, and investigations are called for to see how far this can be reduced.
- (3) Lack of Unanimity of opinion as regards its treatment shews that difficult problems/

problems are to be encountered and that they still await satisfactory solution. The profession is in need of a Mackenzie to advance the lines of treatment of appendicitis.

A short personal account of my difficulties in cases of appendicitis will serve to shew the intricacies of the problem, and will account for my great interest in the subject.

From the excellent teaching I received in my student days at Edinburgh, I gathered that once a diagnosis of acute appendicitis had been made, operative interference should be undertaken forthwith - and the sooner the better. After graduating, I became Assistant in a large colliery practice with poor hospital accommodation, the nearest hospital being twenty miles distant. I well remember my first case of acute appendicitis which occurred in a man twenty-four years of age. I reported the circumstances to my chief, and suggested removal to Cardiff Infirmary for immediate operation. My chief saw the case, agreed with the diagnosis, but advised medical treatment. This consisted in keeping the patient in the Fowler position, and allowing only sips of water by mouth. To my surprise, this and ten or twelve subsequent cases of acute appendicitis treated along similar lines, made an uninterrupted recovery. I was now in a dilemma as to what treatment should be adopted in these cases. While I had been instructed/

instructed as an undergraduate that immediate operation was advisable, my chief (M.D., F.R.C.S.E.) had demonstrated that rather than submit such cases to the ordeal of a long, jolting journey, it was better in the majority of cases to institute "medical treatment" until the attack had subsided, appendicectomy being performed in the quiescent period. Adopting the latter line of treatment, I found that several cases of acute appendicitis responded very well indeed; one patient, however, a boy age 12, so far from improving under the régime, was definitely becoming worse. He was sent to Cardiff Infirmary and submitted to immediate operation. A perforated gangrenous appendix with spreading peritonitis was found, but fortunately the patient recovered.

This proved to me that while some cases of acute appendicitis will recover without operation, there are others which require immediate operation. I set myself the task of finding out why some recover and why others go on to peritonitis.

At this time a Cottage Hospital was opened, to which my chief was appointed Surgeon. We were now in the fortunate position of being able to observe the earliest manifestations of the disease in our patients, and to operate immediately if necessary.

We could also note the pathological state of the organ, and supervise the post-operative care of the patient.

I was now able to make observations regarding the relationship between the clinical symptoms and the pathological findings as revealed at operation.

In my present appointment as 1st Assistant Medical Officer in a Poor Law General Hospital, I examine many cases of appendicitis sent in by local practitioners and have had an opportunity of operating upon large numbers of them.

I have thus observed the treatment of cases of appendicitis from three viewpoints -

- (1) In general practice, with poor hospital accommodation.
- (2) In general practice with an attached hospital where the cases could be seen in their inception, operated upon if necessary, and the effects noted.
- (3) In a surgical hospital, where the patients were seen often with the disease well established.

I believe I have thus had an excellent opportunity of acquiring a composite picture of the clinical aspects of appendicitis, from its earliest inception to its most advanced stages. The results of this experience, together with the result of my investigation into the factors which will allow one type of appendicitis to recover without operation, and the factors which will determine the development of general peritonitis in another, I hope to embody in this present thesis, which is based on an analysis of 600 cases.

I am indebted to Mr. Richard Muir of the Pathology/

Pathology Department of Edinburgh University for his excellent coloured reproductions of two specimens.

A separate atlas contains photographs, and sections of appendices etc. with attached reports.

CHAPTER I.

A N A T O M Y

The vermiform appendix is a round worm-like structure situated in the abdomen and lies near the junction of the small and large intestines. It is attached by its base to the postero-medial wall of the caecum, while its free rounded extremity or tip may point in various directions. It varies greatly in dimensions but is on an average 3 - 4 inches long and $\frac{1}{4}$ inch wide.

Considerable surgical importance is attached to the actual position the organ occupies and this is determined (1) by a consideration of the Embryological Development and Rotation of the Intestines. (2) by a consideration of the Topography of the Right Iliac Fossa.

EMBRYOLOGY

Deaver of Philadelphia states that there is at first no caecum present in the embryological development of the human intestinal tract. "The original tract consists of a straight tube and is divided into the fore gut, mid gut and hind gut. It is attached to the umbilicus by the vitelline duct which sometimes persists in the adult as Meckel's diverticulum. This attachment of the gut to the anterior abdominal wall pulls the formerly straight tube into a U-shaped projection, consisting of an upper/

upper and lower limb. The caecum is distinct at the end of the sixth week of intra-uterine life; it buds from the lower limb of the primitive intestinal loop and locates the division between the small and large intestine. As development proceeds the caecum increases in length, and from its lower extremity the vermiform appendix appears as a tapering point. The lower limb of the loop then ascends across the upper, past the umbilicus to the left hypochondrium, thence across to the right hypochondrium and finally descends and, in the vast majority of instances, reaches the right iliac region about the end of the sixth month although even at birth and in early childhood the caecum is placed higher than in adults."

Dott of Edinburgh has recently written a masterly article on "Anomalies of Intestinal Rotation" in which he shews that the above rotation may be arrested at different stages, and that the caecum with its attached appendix may assume different anatomical positions within the abdomen commensurate with the various degrees of failure of development and rotation. For example, the caecum and appendix may be found in the region of the gall bladder, or in the left hypochondrium, and he states that "in cases of obscure inflammatory lesions of the left side of the abdomen, appendicitis with non-rotation of the intestine is to be considered -- more especially in/

in the young." These abnormalities of position are important and ought to be kept in mind in the diagnosis and treatment of appendicitis for, as Dott says (loc. cit.) "has not left sided appendicitis been kept under observation with disastrous consequences when a normally placed organ would have been removed by timely operation?"

It will be seen from the above that the vermiform appendix is usually situated in the right-iliac fossa but that, due to developmental abnormalities, it may be found in aberrant situations, chiefly the left iliac fossa, right hypochondrium in association with the liver and gall bladder, or in the region of the right kidney.

We now proceed to consider the different positions it may be stated to assume in the right iliac fossa, and this presupposes a knowledge of the topography of the right iliac region.

TOPOGRAPHY OF RIGHT ILIAC REGION

There are four factors which will determine the actual position of the appendix in the right iliac fossa.

- (1) Relation of appendix to the caecum and posterior abdominal wall.
- (2) Mesentery of the appendix.
- (3) Folds.
- (4) Fossæ of the peritoneum in relation to the appendix.

1. Relation to the caecum.— Berry has shewn that/

that the vermiform appendix of man is represented in the vertebrate kingdom by a mass of lymphoid tissue situated most frequently at the caecal apex. In the so-called foetal type of caecum in man this relationship of appendix to caecum still persists, the appendix being the direct continuation of the caecal apex. But it is so much more frequent to find the base of the vermiform appendix attached to the postero medial wall of the caecum just below the ileo-caecal valve. This is due to the greater embryological development of the outer and anterior walls of the caecum causing the base of the appendix to be displaced medially and posteriorly.

The opening of the base of the appendix into the caecum is guarded by an oval fold of mucous membrane - the so-called valve of Gerlach. This has been regarded by some as controlling the passage of contents from caecum to appendix and vice versa, and is considered as having an important bearing on the aetiology of appendicitis. Kelly, however, states that "this structure is not a valve in the true sense of the word, and is clearly seen only when the appendix comes off at an acute angle from the caecum." Again, Sir Frederick Treves in his work on "Perityphlitis" says "The valve of Gerlach which was supposed to protect the orifice of the appendix has no existence."

A point of the greatest practical importance in the/

the surgery of the appendix depends on the relation of the appendix to the caecum and to the posterior abdominal wall. If, during the descent of the caecum in the third stage of rotation of the intestine the peritoneum covering the posterior wall of the caecum fuses with the posterior abdominal wall, a retro-caecal appendix will be extra-peritoneal. Consequently a diseased appendix in this position can be removed retro-peritoneally without exposing the peritoneal cavity to the dangers of infection and contamination. If, however, the fusion is not complete, a retro-caecal appendix may still be intra-peritoneal and must be removed by the peritoneal route. It can readily be seen that the above relationship is of immense practical importance.

2. Mesentery of the Appendix.— Although the appendix is covered on all sides by peritoneum which forms its serous coat, it has also attached to it a double fold of peritoneum which is derived from the left or lower layer of the mesentery of the ileum. To this double fold is applied the name mesentery of the appendix, meso-appendix, or mesenteriolum. It is quadrilateral in shape - the four sides being formed by (a) the continuation of the left portion of the ileal mesentery (b) the free border (c) the attachment to the appendix (d) the junction with the caecum (Royster). It usually extends to the tip of the appendix but may only extend half way or less and/

and I have seen one case post-mortem where there was no meso-appendix — the appendix being entirely retro-peritoneal.

The length of the mesentery of the appendix will affect the position and mobility of the appendix. Thus a long mesentery will allow the appendix to become pelvic in position and will predispose to spiral kinking of the appendix upon itself. Several cases of volvulus of the appendix upon the mesentery have been reported in the literature and were attributed to undue mobility of the organ. A short mesentery will pull the appendix in an upward direction either in front of, or behind the terminal ileum.

Contained within the two folds of the mesentery are lymphatics, nerves and glands, while the free border transmits the artery and vein of the appendix. As the artery is an "end-artery" torsion of the meso-appendix will result in gangrene of the appendix due to obliteration of the blood supply.

3. & 4. Folds and Fossæ of the ileo caecal region

Various peritoneal folds have been described around the caecum and appendix, considerable work having been done on this subject by Lockwood & Rolleston who made frozen sections of post-mortem specimens. These folds are described in different ways by different observers and are named after their advocates, e.g., the bloodless fold of Treves, the mesenterico-parietal/

mesenterico-parietal fold of Jonnesco, the pericolic membrane of Jackson, Clado's ligament, Lane's kink, etc.

These folds enclose fossæ which in turn are designated by different names. The appendix may be ensnared in these fossæ. We shall quote Kelly's description which will be more easily understood by referring to Diagram (p. 1) in the accompanying atlas. He divides the folds and fossæ about the ileo-cæcal region into the peri-cæcal, and the retro-cæcal or subcæcal.

"The peri-cæcal folds lie above and to the left of the cæcum and include the ileo-colic and ileo-cæcal folds and are, with a very few exceptions, constant. By retro-cæcal or subcæcal folds and fossæ we understand those situated behind and beneath the cæcum; while the former are at once visible, the latter cannot be seen without lifting up the cæcum.

The ileo-colic fold lies in the angle between the ileum and the colon. It is semilunar in shape, its free or concave margin being turned toward the ileum. Along its free or inner margin the anterior ileo-colic artery passes with its accompanying vein; also several lymphatic channels. Frequently it contains one or more lymphatic glands embedded between its two peritoneal layers, also a varying amount of fat. The ileo-colic fold covers the ileo-colic fossa; the/

the floor of this fossa is formed by the ileum and colon at their junction, and by their mesentery.

The ileo-caecal fold occupies the ileo-caecal angle and lies anterior to the meso-appendix. Its superior border is attached to the lower edge of the ileum for a distance of 5 to 10 cms. Its right or external border lies along the caecum; its inferior border becomes lost on the anterior surface of the meso-appendix, while the internal border is free and concave and forms the entrance to the ileo-caecal fossa, which varies in depth and size according to the size of the fold.

The retro-caecal or subcaecal folds and fossæ depend entirely for their existence upon the coalescence or adhesion of the colon, caecum and mesentery to the posterior abdominal wall, and are therefore secondary in origin as compared with the folds previously described. In some cases, the caecum never becomes adherent to the posterior abdominal wall, and we therefore find no retro-caecal folds or fossæ.

The most capacious and the most constant of the fossæ is the internal retro-colic (See Atlas p. 1) also called the inferior ileo-caecal, which can only be demonstrated by lifting up the caecum, appendix and ileum. It is then seen as a funnel-shaped pocket extending in an upward direction under the ileum and colon. From the depth of this fossa arises the posterior leaf of meso-appendix, and the entire/

entire appendix is often found curled up in this space, while its tip may point in various directions."

Before proceeding to the description of the Position of the Appendix, reference must be made to a rather important fold, the "genito-mesenteric fold of Reid". Gladstone & Wakeley describe this as "being usually triangular, having an anterior surface directed forward and to the left, a posterior directed backwards and to the right. Of the three borders, two are fixed and one is free. Thus, there is usually a posterior fixed border attached to the posterior abdominal wall and extending from the duodenal region downwards on the right psoas muscle to the pelvis; an upper attached border connected with the under surface of the mesentery of the small intestine near the ileo-caecal orifice or with the ileum in the same situation and a free border usually directed forward and to the right. The lower end of the fold in the female passes over the brim of the pelvis, on to the posterior aspect of the broad ligament of the uterus, and is frequently blended with the suspensory ligament of the ovary. In the male, it may end in the neighbourhood of the internal abdominal ring, or pass over the brim of the pelvis to the lower end of the fold raised by the obliterated hypogastric artery."

Reid believes that this band by its traction on the ileum is responsible for the descent of the caecum/

caecum and appendix into the right iliac fossa. It is also of importance in that it may lead to constriction of the terminal ileum and I believe will possibly explain the occurrence of the appendix in hernial sacs in the male and the association of ovaritis and appendicitis in the female.

Wilkie regards this fold as being important for two reasons. He states that it frequently binds the middle third of the appendix downwards towards the pelvic brim and predisposes to acute appendicular obstruction. Again, in his recent "Murphy Oration" he states that "the usual impediment to easy removal of the appendix is not the presence of inflammatory adhesions but the bending down of the middle third of the organ by that congenital fold, first described by Douglas Reid as the genito mesenteric fold. This fold is practically bloodless, and a few touches of the knife, dividing it, will mobilise the appendix completely and allow it to come up without tension into the abdominal wound."

This ligament would seem to be somewhat similar to that described by Clado in 1892; Clado's ligament, as it is called, is a fold of peritoneum extending from the right broad ligament to the appendix. Its presence is challenged by some, but is held by others to form a possible source of communication between the appendix and ovary, both as regards lymphatics and blood supply. It would appear to be a portion of/

of the genito-mesenteric fold of Reid.

POSITION OF THE APPENDIX

The vermiform appendix is usually situated in the right iliac fossa, and although the attachment of its base to the caecum is fairly constant, its terminal portion may point in any direction, and its general position may vary within wide limits.

Various classifications for the description of these positions have been adopted, with much resultant confusion, e.g., Bristow classifies the positions according to the compass direction of the appendix from a central fixed point in the right iliac fossa which represents the base of the appendix. Barnard describes the position in terms of the hands of a clock, while others use the terms "pelvic", "retro-caecal", "mesenteric", etc. A diagram in the accompanying atlas (p. 2) shews the various possible positions, and follows Kelly's classification.

A more recent one has been made by Gladstone & Wakeley: according to these observers, the appendix may assume the following positions :-

1. Post-caecal or retro-colic
2. Pelvic or psoas, near or hanging over the brim.
3. Sub-caecal
4. Anterior or pre-ileal
5. "Splenic" or post-ileal
6. Ectopic.

They/

They have made two reports of the frequency of these positions as revealed by examination at operation, at post-mortem, and in the dissecting rooms. The first was based on an examination of 3,000 cases, the second on 5,000 cases. Their results may be tabulated thus -

Position	Percentage in 3,000	Percentage in 5,000
Post-cæcal and retro-colic	69.2	64.38
Pelvic or psoas	27.5	32.11
Sub-caecal	1.8	2.02
Anterior or pre-ileal ..	0.9	0.94
Splenic or post-ileal ..	0.5	0.5
Ectopic ..	0.036	0.04

It will be seen from the above that the most common position is the post-caecal appendix, and according to the above observers, this type of appendix may be found

- (a) free in a post-caecal or retro-colic pouch of peritoneum.
- (b) held in contact with the caecum or ascending colon by a short mesentery.
- (c) adherent to the caecum, or colon, which with the appendix forms the anterior wall of a retro-colic pouch of peritoneum.
- (d) behind the caecum and ascending colon, but owing to the obliteration of the retro-colic pouch entirely extra-peritoneal.

HISTOLOGY OF THE APPENDIX

The appendix lying as it does at the junction of the small and large intestines, seems to be a histological hybrid of them. It resembles the small intestine in the richness of its lymphoid tissue, and the disposition of its muscular coat; while the absence of villi and valvulae conniventes makes it more akin in structure to the large gut. A study of the transverse section shewed in p.4(i) of the Atlas will shew that it is possessed of four coats:

- (1) Peritoneal or Serous Coat - This is composed of flat endothelial cells and is continuous with the meso-appendix.
- (2) Muscular Coat which consists of
 - (a) an outer layer of longitudinal muscle and
 - (b) an inner layer of circular muscle.

Lockwood has shewn that there are areas where, due to gaps in the continuity of the muscular coat, the submucous and peritoneal coats become contiguous. Through this hiatus muscularis (as Lockwood calls it) blood vessels pass from the meso-appendix to the mucosa; Lockwood would also regard "this continuity of submucosa and subperitoneal tissue as being of great pathological importance, as it is the chief road by which the infective inflammations of the mucosa reach the peritoneum."

I believe that this relationship of blood vessels to muscular coat has a significance hitherto unappreciated. It is my conviction that the administration of purgatives is the commonest cause of precipitating perforation and gangrene in acute appendicitis; purgatives by causing spasm produce compression of the blood vessels as they pass through the hiatus muscularis. Ischaemia of the mucous and submucous coats results, and if/

if infection be present, predisposes to onset of gangrene.

The hiatus muscularis would appear to be of great pathological significance.

- (3) Submucous Coat - This is largely composed of fat, but has also a rich supply of blood vessels, lymphatics and nerves. It is this coat which is chiefly thickened in chronic appendicitis. It must be pointed that the submucosa is absent in children.
- (4) Mucous Coat - This consists of
- (a) a single layer of columnar epithelium
 - (b) numerous tubular glands opening on to the lumen as the crypts of Lieberkuhn. These glands are entirely confined to the mucosa and do not extend beyond the muscularis mucosae.
 - (c) the muscularis mucosae, which is often absent, is a thin band of muscle separating mucosa from submucosa.
 - (d) lymphoid follicles - these are oval in shape, and although chiefly found in the mucosa, a few penetrate the muscularis mucosae to lie in the submucosa. Their periphery stains more deeply than their centre, and the average number in a transverse section is 5 or 6.

Berry - from research carried out in the Royal College of Physicians Laboratory, Edinburgh as regards the lymphoid tissue of the appendix - tabulated the average number of lymphoid follicles present in a single transverse section through the centre of the human appendix, as follows :-

Below 1 year	...	5	...
1 -10 years	...	6	...
10 -20 "	...	7	...
20 -30 "	...	6	...
30 -40 "	...	3	...
40/			

40 - 50 years	...	3	...
50 - 60 "	...	2	...
60 - 70 "	...	trace only	
70 - 80 "	...	practically none.	

He concludes that the appendix is an actively functioning gland until the fourth decade.

It is interesting to note that the highest incidence of appendicitis is from 10th-20th year - when, according to Berry, the lymphoid tissue is most abundant.

It is estimated that the total number of lymphoid follicles present in an appendix ranges between 150 - 200 (Lockwood), a fact which led Sir John Bland-Sutton to refer to the appendix as the "abdominal tonsil."

BLOOD SUPPLY OF APPENDIX

The appendix, being a portion of the mid-gut, receives its blood supply from the superior mesenteric artery (Dott). Its posterior ileo-caecal branch forms the chief blood supply of the appendix, and runs down behind the ileum, to give off appendicular branches which run in the free border of the meso-appendix. It must be emphasized that the appendicular artery is an "end-artery"; hence gangrene will readily result from interference with blood supply, be it either mechanical or inflammatory in origin.

Some hold that there is an additional blood supply in the female via the ligament of Clado, and Backmann considers this the explanation of the fact that "there is a much smaller proportion of destructive appendicitis in the female than in the male."

VENOUS RETURN

The veins of the appendix take origin from the capillaries in its walls and unite in the meso-appendix to form the appendicular vein. This joins the ileo-colic vein which ultimately empties into the portal vein. This anatomical fact explains the occurrence of pylephlebitis in certain cases of acute appendicitis; and to prevent this complication, Braun makes a practice of ligating the ileo-colic vein in all cases of gangrenous appendicitis.

LYMPHATIC SUPPLY

Lymphatic vessels pass from the basilar lymph sinus around the lymphoid follicles through the hiatus muscularis of Lockwood to a lymphatic ganglion in the ileo-caecal angle. There is also a communication in the female via the ligament of Clado with the lymphatics of the pelvis and may explain the co-existence of appendicitis and what I like to term "appendagitis," i.e. inflammation of the ovary and tube.

Recent experimental work by Braithwaite has thrown considerable light on the relationship between appendicitis and gastric and duodenal ulcer and cholecystitis. He states that "the normal flow of lymph from the ileo-caecal angle in a perfectly healthy individual, as gleaned from post-mortem injections - largely in the foetus - is clearly defined. Gathering tributaries from the appendix, the caecum and/

and the last six inches of the small intestine (in part only), the lymphatics pass irregularly in a group of five or six vessels to the chain of glands which lies dotted along the line of the ileo-colic artery. The main afferent trunks pass up in front of the third part of the duodenum, reaching the group of glands massed round the superior mesenteric artery, and so to the lumbar chain."

As a result of experiments with dyes on animals and human beings he concluded that thence "most of the lymph undoubtedly passes deeply to join the lumbar group of glands; but in living subjects some is seen to pass upwards over the head of the pancreas to enter that group of glands which frequently lies in a crescent, or in a series of small groups along the inner border of the curled duodenum." He also states that "some lymph undoubtedly passes through this group on to the duodenal wall itself and up to and occasionally beyond the pylorus."

He adduces evidence to support his belief that duodenal and gastric ulcer may result by a retrograde flow of infected lymph from the appendix to the wall of the duodenum or stomach because of obstruction of the normal lymph flow consequent upon lymphangitis and lymphadenitis; thus "the infected lymph (e.g. from an acute appendicitis), failing to make its usual exit, seeks new avenues of escape in all directions; misses whole groups of glands which, under ordinary conditions would check and filter it; and ebbs/

ebbs and flows to and fro until the glands around the superior mesenteric artery are reached and partly obstructed. Deprived of easy access through them to the lumbar glands, part of the lymph flows onwards over the head of the pancreas and enters the glands on the concavity of the duodenum, and in process of time bathes even the duodenal wall itself, escaping finally to the coeliac glands by normal or possibly aberrant paths, and so reaches the receptaculum chyli. Is it not a reasonable suggestion that similar ineffective lymph may pass along the great omentum, and through a similar process of obstruction of the gastroepiploic glands - reach the stomach?" He believes the bathing of the duodenal and gastric mucosa by such infected lymph to be the starting point of duodenal and gastric ulceration.

Wilkie of Edinburgh, as we shall see later, believes this association of peptic ulcer and appendicitis to be due rather to retrograde venous emboli from omental veins.

Costain, believing that toxins arising from diseased appendices reached the general circulation via the thoracic duct, advocated the operation of thoracostomy in cases of appendicitis with general peritonitis. Paterson Brown of Edinburgh, however, has recently adduced from experimental investigation on rabbits that this operation holds out little prospect of elimination of toxic absorption in man.

NERVE/

NERVE SUPPLY

The appendix derives its nerve supply from the superior mesenteric plexus of the sympathetic, the branches accompanying the ileo-colic artery to reach the appendix.

The segment of the spinal cord from which the nerves to the appendix are derived are the same as that from whence arise the 11th and 12th thoracic nerves. This fact is of importance in explaining the areas of tenderness and rigidity in diseases of the appendix.

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CHAPTER II.PHYSIOLOGYOF THEAPPENDIX

This leads us to consider the functional importance of the appendix in the animal economy. Views of the utmost diversity have been advanced by the most eminent members of the profession, and we shall very briefly mention, without comment, their statements and opinions.

The functional importance of the appendix - like that of any organ - may be assessed from a consideration of three things.

- (1) Its phylogeny and development.
- (2) Its structure (as structure and function are inter-related).
- (3) The effects of its removal.

(1) It is a fact that the vermiform appendix in man is much smaller than that found in the herbivora, and Metchnikoff regards the whole of the colon together with the appendix in man as useless or vestigial structures; the appendix he regards as suffering from disuse atrophy.

Keith refutes this opinion, and states that it is hardly correct to regard a structure as vestigial which is present in all at birth, and which does not attain/

attain its maximum length until 40 years of age. He suggests the appendix is suffering from what he terms "abiotrophy" - i.e. presenile atrophy.

Berry, too, strongly supports the view that the appendix is a useful organ: from a comparative study of the "caecal apex" in animals he concludes that the vermiform appendix represents the "caecal apex" of the vertebrate kingdom; it is not a vestigial structure, but is a specialised part of the alimentary canal.

(2) Structure. — Different observers would ascribe specialised functions to the histological elements found in the appendix, e.g.,

As regards the Mucous Membrane.— Dudgeon and Mitchiner regard the epithelium as being concerned in fat absorption. Macewen, of Glasgow, believed the glands, by secreting mucus, "exercised a controlling influence on the bacteria within the bowel."

Sir Arthur Keith would regard the caecum as a second stomach, and he states that "all food has to pass through the appendix for complete caecal digestion."

Heile, as a result of experiments on dogs, attributes a digestive function to the appendix, the glands secreting ferments which split albumen and carbohydrate.

As regards Lymphoid Tissue.— Sir John Bland-Sutton referred to the appendix as the "abdominal tonsil", and/

and many observers hold that invasion of the appendix by micro-organisms results because of the richness of lymphoid tissue.

Rosenow reminds us that the appendix, apart from the tonsils, is the portion of the alimentary canal most richly supplied with lymphoid tissue.

K. H. Digny would regard this as a beneficial factor, and would assign to the appendix an important function of "protecting the body against chance infection by means of a continuous process of auto-vaccination."

As regards musculature.— Adami and Macrae state that the appendix by its musculature initiates the peristalsis of the large gut, a view supported by Heile who regards the musculature of the ileo-caecal region, including the appendix, as forming a single whole in its muscular action, each element having its share in effectual peristalsis.

(3) Effects of Removal.— Binet and Dubois; Heile, and others, from a study of the effects of removal of the organ, even suggest that the appendix has an internal secretion, and that its removal leads to obesity. But surely it can be argued that this obesity may result from improved digestion, following removal of a pathological appendix.

Summing up, it will be seen that some would regard the appendix as being quite important and not lightly to be removed; others more sceptical would regard/

regard it as being retrogressive and of little importance. One humorous surgeon suggests that its chief function is "to contribute largely to the upkeep of a noble profession." We can only agree with Deaver of Philadelphia when he says "these opposing views are not necessarily dissonant. We can believe that the appendix has a function, yet it is essentially vestigial in character, and that its potentialities for harm are greater than its capability for good."

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CHAPTER III.

A E T I O L O G Y.

The aetiology of appendicitis like that of any other disease may be considered under the two headings -

- (1) Predisposing causes and
- (2) Actual cause.

Thereafter it may not be inappropriate to consider the pathogenesis of the condition, i.e., the modus operandi of the factors concerned.

1. PREDISPOSING CAUSES

These may be subdivided into two groups -

- (a) General factors e.g., geographical distribution, season, climate, family susceptibility, age, sex, diet, constipation.
- (b) Factors relating to the appendix itself including consideration of anatomical position and the peculiarities of its structure.

(a) General factors:

Geographical distribution.— Appendicitis is one of the prices of civilisation and is especially frequent in temperate zones. Rendle Short of Bristol has recently written an exhaustive treatise on this aspect of the disease in which he shows "that appendicitis is common in the more civilised European and American countries and very rare in Asiatics, Africans, and Polynesians. If, however, individuals from these countries are taken into the service or society of Europeans/

Europeans and eat their food, they acquire the European's liability to the disease. He states a curious and interesting fact, viz. that apes in captivity acquire the human liability to appendicitis". The high frequency of the disease in America has been attributed to the speeding up of modern civilisation with its concomitants of hurried meals and the non-observance of nature's call to stool.

Season and climate.— It is undoubtedly true that the disease would seem to occur in epidemics, being most common in the spring and early autumn, and is often associated with acute tonsillitis, rheumatism and influenza.

While in General Practice, I was struck with the close relationship of appendicitis to these aforementioned diseases. Riff even goes so far as to suggest that the disease is contagious.

Family susceptibility.— In taking the clinical history in cases of appendicitis, we have observed the disease to have a predilection for certain families, e.g., I have notes of one family of eleven, all of whom suffered from constipation and all of whom underwent operations for appendicitis.

Again a few months ago, while a patient was convalescing after an operation for acute appendicitis, his son aged five years was admitted to hospital suffering from general peritonitis due to a perforated appendix. That these occurrences are not merely fortuitous/

fortuitous is borne out by similar experience of more competent observers.

Age.— From an analysis of my own cases, it would appear to be most frequent, in the acute stage, between the ages of 10 and 30, and in its chronic stage, between the ages of 25 and 40. No age, however, is immune for I have observed the disease in a child 3 weeks old, and, at the other extreme of life, in a man aged 75. The baby aged 3 weeks was moribund when admitted to hospital and was thought to have a maldevelopment of the intestine; it was only at post-mortem examination that an appendix abscess was found.

It is interesting to note that the highest incidence of acute appendicitis occurs at an age, when-- according to Berry -- the appendix is most richly endowed with lymphoid follicles.

Sex.— My experience shows a slight preponderance in the male sex and this lesser frequency in females has been attributed to (a) a lower meat consumption and (b) additional blood supply to the appendix via the ligament of Clado.

Diet.— Statistical and experimental evidence has been brought forward to show that the consumption of meat is an important predisposing factor. Short (loc. cit.) presents statistics which prove that periods of high consumption of imported beef correspond with high incidence of the disease; and "Institutions/

"Institutions where the older and cheaper dietary prevails enjoy a relative immunity."

Lucas Championnière makes a similar assertion. He states that appendicitis is confined chiefly to countries where meat forms a large part of the people's food and that it is invariably most severe in those who eat meat in excess while vegetarians are almost free from it. Interesting statistics covering many countries and years are offered as proof. In every section of France, the frequency of appendicitis increases in direct ratio to the increased consumption of meat. In convents, schools, prisons, asylums, where the diet is almost or even entirely vegetarian, cases of appendicitis are very rare. In one prison with a thousand prisoners only one case occurred in four years. In an asylum with 1,500 inmates not one case occurred in three years. In Rumania one case of appendicitis was noted among 22,000 sick persons of a vegetarian population; while in meat consumers one case occurred among 221 sick persons. Recent letters in the British Medical Journal have also emphasized the relative immunity of the vegetarian; the simpler the diet, the less apparently the liability to attack.

The above views find a counterpart in the interesting experimental work of D.P.D. Wilkie of Edinburgh. He experimented on cats and, as they do not possess an appendix, he furnished them with an artificial/

artificial one by isolating the last coil of ileum, which is rich in lymphoid tissue, and restoring the continuity of the intestinal tract. "The behaviour of this isolated coil of ileum was then observed (1) when empty; (2) when containing a small quantity of faecal matter; (3) when more or less full of faecal matter. The empty loop formed a mucocoele; the loop containing a small amount of faeces, an empyema, which eventually ruptured if left; whilst the loop containing a considerable quantity of faecal matter became rapidly gangrenous. When the loop was filled with an emulsion of intestinal bacteria grown on culture, no striking changes supervened although in some cases it slowly filled with a muco-purulent content.

Decomposition of faecal matter appeared to be the fact inducing the rapid pathological changes met with in the group of rapidly fatal cases. It was found, moreover, that the gangrene was more rapid and pronounced if the animals had previously been fed on a rich proteid diet than if they had been given a more purely carbohydrate diet."

Constipation.— I believe this is of great ætiological importance. Most patients suffering from appendicitis give a history of constipation; and stasis of contents as usual predisposes to infection. The immunity of the vegetarian could be explained as being due to the less likelihood of constipation./

constipation. Again, appendicitis is more common in civilised countries because of the greater occurrence of constipation. Prof. Gulland was fond of telling us in his lectures that constipation was responsible for 50% of human illness, and I believe it is one of the most potent causes of the incidence and increased frequency of appendicitis.

(b) Local factors relating to the appendix:

Anatomical position.— Eric J. Ll. Jones-Evans stresses the mechanical factor in the causation of appendicitis. He indicts the comparatively free mesentery containing the chief blood supply because of the rotatory movements it performs on its own axis. "It is my opinion", he states, "that the great majority of inflamed appendices are produced primarily by twisting of the organ, together with its mesentery, with the result that the appendicular artery becomes occluded. The blood supply thus being shut off, infective processes supervene, and the case terminates either in localised abscess formation or, if the appendix ruptures, in general peritonitis. The condition is analogous to strangulated hernia, a twisted pedicle to an ovarian cyst, or torsion of the spermatic cord." Recurrent appendicitis he explains on the assumption that the torsion has not been sufficient to cut off completely the blood supply.

The importance of the genito-mesenteric fold of Reid by preventing the appendix from emptying itself in/

in the causation of appendicitis has already been alluded to.

Structural peculiarities.— The appendix being a retrogressive organ is liable, as Darwin has pointed out, to show a congenital lack of the power of resistance and is frequently the seat of disease. Again, its wealth of lymphoid tissue with its "end" blood supply makes it a locus minoris resistentiae and an easy prey to invasion by micro-organisms.

2. ACTUAL CAUSE

There can be no doubt that appendicitis is the result of bacterial invasion. Various germs have been held responsible and it would appear that, as in broncho-pneumonia, there is no specific micro-organism. The streptococcus is usually regarded as the commonest primary organism with the Bacillus coli communis as a secondary invader.

The chief workers on the bacteriology of appendicitis are Tavel and Lanz; Dudgeon and Sargent; Rosenow, and Dudgeon & Mitchiner.

It has already been shewn that certain peculiarities of structure in the appendix make it a site of lowered resistance. Again, the virulence of micro-organisms may be increased by stasis consequent upon constipation or inability of the appendix itself due to kinks or angulations. This lowered resistance of tissue and exaltation of virulence of micro-organisms make infection abnormally frequent.

Foreign Bodies:

Happily the day is long past when cherry stones, orange seeds and buttons were looked upon as the actual cause of appendicitis. But it seems difficult even now to assess the aetiological importance of faecoliths and intestinal parasites. Faecoliths have been found in 0% to 10% of inflamed appendices by different writers. The relationship is perhaps a dual one. Mild attacks of appendicitis may lead to stasis of faecal content and a faecolith be formed. The presence of this faecolith will predispose to further infection. Diverse opinions are held as regards the importance of intestinal parasites. Rendle Short of Bristol states that on fifteen occasions he made a diagnosis of acute febrile appendicitis. He removed an organ apparently normal but inspection showed that it contained thread worms. "One will usually find", he says, "the nearest lymphatic nodes swollen in these cases, and it seems beyond question that the pain, fever, and vomiting are due to the presence of worms."

Riff brings forward the following arguments in favour of appendicitis being due to oxyurides, (1) Appendicitis affects all classes of society alike (2) it is most frequent between 5 and 20 - a period of life during which oxyurides are most prevalent. (3) It is very rare in the infant in spite of the frequency of intestinal affections at this age. The relative/

relative immunity of the child is due to its being kept clean. (4) Appendicitis is contagious and may affect several members of the same family. In 152 appendices studied at Strasburg before the War and presenting all forms of acute and chronic inflammation, he found oxyurides in 48%. Since the War, he has examined 73 more appendices removed at the Hôpital Trousseau in Paris and found the parasites present in 80%. The fact that some observers have never found oxyurides present in the appendix in a large number of cases is attributed by him to the examination having been made after the appendix had been opened by the surgeon and its contents expressed.

Still, of London, found the vermiform appendix to be a common habitat of thread worms. "In 200 autopsies on children under twelve years of age, thread worms were present in the intestines in 38, or 19 per cent., and in those children over twelve years of age the percentage was much higher, viz., 32 per cent. In no less than 25 out of 38 cases the worms were found in the appendix, and in 6 the appendix was the only part of the alimentary canal where the worms were found. In one case where pain had been complained of in the right iliac fossa, the appendix contained 111 worms, and was in a catarrhal condition. In several other cases the appendix was in a similar condition.

Läiven & Reinhardt examined 620 appendices, and found/

found oxyurides present in 60 cases or 9-76 per cent. The worms were found twice as frequently in the female as in the male appendix; 12 of the affected appendices occurred in children up to the age of 14, and 48 in adults. They regard the condition as being due to penetration of the mucous membrane by the oxyurides, thus opening the path for bacterial invasion. It is probable that the toxic action of the parasites causes a superficial transient inflammation of the mucous membrane. Clinically, a certain diagnosis cannot be made, but relatively slight objective findings, combined with fairly acute or chronic symptoms is suggestive of oxyuris appendicitis.

Becker found oxyurides in 50 per cent. of all normal appendices surgically removed. In appendices surgically removed in consequence of acute appendicitis, oxyuris was found in the same proportions as in those obtained at the post-mortem table. He regards an aetiological connection as being excluded.

Eastwood examined 76 appendices, and found no evidence in support of the theory that oxyuris vermicularis is a frequent cause of appendicitis. The parasite occurred in about the same proportion of cases of appendicitis and of normal appendices post-mortem. No local eosinophilia was found when the worm was present in the appendix. Eosinophil cells were found in the mucosa of the appendix in much greater numbers in inflammation than when the part was/

was normal.

as

Brauch regards true appendicitis/arising from bacterial enterogenous infection of the mucous membrane of the appendix. Thread-worms, he regards, as being casual findings unconnected with recent or old inflammatory changes; supposed "worm-canals" of the mucosa are merely artefacts.

An interesting discussion on the relationship of worms to appendicitis is reported in Edinburgh Medical Journal (1913); 10; 437.

In my own series, I have observed thread-worms in only three cases. In the lumen of specimen No. 6, a female oxyuris vermicularis can be seen, and is recognised by the characteristic ova contained in its ovarian tubes.

PATHOGENESIS.

The question as to whether or not any tissue or organ will suffer the inroads of bacteria must ultimately depend upon the relationship between the resistance of the tissues or organ and the virulence of the attacking micro-organism. If the former be high, and the latter attenuated, good health is enjoyed; but if the tissues have a lowered resistance, and the invading organism an increased virulence, inflammatory changes are prone to result. These latter conditions obtain in the vermiform appendix.

That there is a lowered resistance is consequent upon the histological peculiarities we have already noted in the appendix, viz. -

- (1) Retrogressive organ.
- (2) Excess of lymphoid tissue.
- (3) End artery.

And exaltation of virulence of organism in a blind tube has been regarded to sum up the entire history of appendicitis (Dieulafoy).

This exaltation in virulence will result from stasis of contents, e.g., constipation; kinking of organ by genito-mesenteric fold; stenosis following previous inflammation; obstruction by fæcolith, etc. The highest age incidence corresponds to the period of greatest wealth of lymphoid tissue (Berry), and Wilkie gives two reasons for the greater frequency in males, "First, the adolescent and young adult male partakes/

partakes of a diet which is considerably richer in protein than does the female of corresponding age; and, secondly, he is more exposed to strain and injury such as might determine the entrance of faecal matter to an appendix with a stenosis or kink near its proximal end."

It still remains to discuss the path of entry of the invading organism. Until recently it was assumed that infection proceeded from the lumen but the important work of Rosenow on elective affinity of micro-organisms suggests that it is a blood borne infection. Rosenow concluded from experimental investigation on animals that many inflammations of organs, e.g., the appendix, stomach, pancreas, gall-bladder, hitherto regarded as due to infection from neighbouring surfaces, may really be blood borne from some distant foci. In favour of this view, he presents the following arguments.

(1) The early lesions of these organs are often sharply defined, hæmorrhagic and situated in any of the coats, without necessary involvement of the mucous membrane.

(2) He has caused infection of the pancreas by intravenous injection of bacteria, and found them localised chiefly in the pancreatic head, just as much as in the usual clinical cases in which this localisation was supposed to be an indication of spread up the ducts.

(3)/

(3) Experimental feeding of animals with streptococci even when mixed with sharp particles failed to induce gastric ulcer, except in starving animals - yet intravenous injection of these streptococci caused ulceration of the stomach.

Streptococci from acutely diseased appendices when injected into the veins of rabbits, caused appendicular lesions whereas their introduction via the mucous membrane failed to act in this manner, unless the blood supply had been materially damaged. He suggests that in many cases the infection is blood-borne and that the decision as to the site of the damage depends on the elective affinities of the organism travelling in the blood stream but probably originally derived from some distant focus, such as teeth, tonsils, etc. He regards various streptococci as the chief offending organism in these cases, chiefly of the non-haemolytic type and of a low grade of virulence. These varieties are culturally much alike but are differentiated by their behaviour on intravenous injection; they shewed a specific selection in that micro-organisms taken from an inflamed appendix tend to produce appendicular lesions in the injected animals. He states that streptococci found in teeth sockets, tonsillar crypts, etc., although apparently alike, yet possess these elective powers so that one strain may, on injection into the blood stream of animals habitually cause appendicitis, whilst/

whilst another will persistently produce cholecystitis, another gastric ulcer, and so on.

It appears that the bacteria acquire elective localising power in the focus and that the various diseases may be manifestations of phases of the infecting power of the same micro-organism.

This work has been recently substantiated by the remarkable work of A. L. Wilkie, in which he shews that cholecystitis is due to a streptococcal infection of the walls of the gall-bladder and carried by the blood-stream.

Appendicitis would seem to be analogous to the above. It is interesting to note that the human system seems to be suffering from inroads of the streptococcus. The worst inflammations of the appendix, gall-bladder, uterus, tonsil, etc. are streptococcal in origin, and it would appear, according to Rosenow, that the streptococcus is now "manifesting a phase of high infecting power."

The following practical points can be deduced from Rosenow's work.

- (1) The association of appendicitis and tonsillitis is obvious: the tonsil being the primary focus and the appendix the site of the elective affinity.
- (2) Attention to bad teeth and tonsils should diminish the incidence of appendicitis.
- (3) *Apriori*, anti-streptococcal serum should have a beneficial effect in many cases of appendicitis.

Unfortunately this is not my experience. I have been greatly disappointed with its results; and anti-toxin seems to have no control over the toxic effects originated by the streptococcus.

In/

In conclusion, reference must be made to Behan's work. He conducted experiments on rabbits with a view to determining the relative importance of the various aetiological factors in the production of the various forms of appendicitis. He passed ligatures through different coats of the appendix and, by observing the after pathological changes, he formed the following conclusions.

"From our experience on rabbits it was noted that the only time acute inflammatory reactive changes in the appendix occurred was when the lumen of the appendix was entirely obstructed, with strangulation of the mucous and sub-mucous layers and the muscularis. Obstruction to the lumen alone apparently resulted only in a collection of fluid between the mucosa and the muscularis in the sub-mucous space, so that a cyst of the appendix was formed. However, when the obstruction extended beyond the mucosa and included the sub-mucosa and muscularis, a very marked reactive inflammation resulted, and gangrene of the appendix with sepsis and death of the rabbit took place.

The presence of bacteria in the lumen of the appendix is not, in the rabbit, a necessary prelude to inflammation. Gangrene of the appendix may occur even when previous cultures from the lumen of the appendix show no bacterial growth; however, when inflammation had already taken place and the surface of/

of the appendix was inflamed, cultures from the surface of the appendix gave growths of colon bacilli. This inflammation in our own experiments apparently did not primarily result from the thrombotic changes in the blood vessels of the meso appendix. The blood vessels of the meso appendix may be partially blocked, and no pathological change occur in the appendix. The pathological variation or change took place both in the lumen and in the coats of the appendix and led to inflammatory reaction in the walls of the appendix. This reaction was always distal to the obstruction which we had erected.

How closely the above processes may simulate those which occur in the human is problematical. The appendix of the rabbit is larger, longer, and has a slightly different arrangement of the blood supply than has the human appendix. However, clinical observation seems to confirm, at least to a degree, that the above observations may also be applied to man. Obstruction of the appendix may result clinically when a foreign body is present in the appendix and obstructs the lumen at some point where there has been a constriction. Gradual swelling of the mucosa may be sufficient to produce a strangulation of the sub-mucous and the muscularis layers the same as occurs when a ligature or clamp is applied. When this stage is reached gangrene supervenes."

These experiments lend support to Wilkie's views on/

on obstructive appendicitis and form an interesting comparison to his experiments already referred to. It is also interesting to compare Wilkie's views on the production of gangrene with the above: Wilkie says "Complete gangrene of the appendix is a fairly frequent finding at operation and has, by many, been attributed to a thrombosis of the vessels of the meso-appendix. Whilst thrombosis is sometimes present, I am convinced that it is an effect and not the cause of the gangrene."

SUMMARY

Appendicitis is caused by blood-borne streptococcus - primary focus frequently being tonsils and teeth. Its richness of lymphoid tissue explains the age incidence; thrombotic changes in the meso-appendix are regarded by some as resulting from, rather than causing gangrenous changes in the appendix itself.

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Pathology must ever form the basis of our knowledge of the nature, severity, spread and line of treatment of any disease. It constitutes the foundation rock upon which the super-structure of Diagnosis, Prognosis and Treatment are to be built. For as Osler says "Without a sound understanding of things pathological the surgeon will flounder along in an aimless fashion, hitting now the malady and again the patient, he himself not knowing which."

Again, it was from an accurate knowledge of Pathology that the most common cause of "perityphlitis" was seen to originate from the appendix.

Moynihan, with his great gifts of oratory and exposition, has rightly emphasised the greater importance to the surgeon of a knowledge of the "Pathology of the Living" -- the various pathological processes seen at operation -- over those found at post-mortem examination.

Pathology - and the Pathology of the Living - is to be one of the essentials in discussing this disease and I propose to deal with it under the following headings :-

- I. Pathological lesions of the appendix.
- II. Complications (or spread of the disease.)

A very brief reference has already been made to the/

the bacteriology of the condition and the pathogenesis under the heading of Aetiology.

I. PATHOLOGICAL LESIONS

It cannot be too strongly asserted that the pathological changes labelled under the heading "Appendicitis" may range from simple catarrh on the one hand, to suppuration and gangrene on the other. The whole gamut of the changes met with in ordinary inflammation are to be encountered in considering the inflammatory lesions of the appendix.

There are two big subdivisions:-

- A. Acute appendicitis.
 - (1) Catarrhal.
 - (2) Ulcerative.
 - (3) Suppurative.
 - (4) Gangrenous.
- B. Chronic appendicitis.

In types (2), (3), (4) of subdivision A, peritonitis is liable to occur consequent upon perforation.

In chronic appendicitis, kinking, stricture or obliteration of the lumen may occur.

N.B. The pathological changes now to be described are based upon a study of my own specimens and are illustrated in the accompanying Atlas.



A. ACUTE APPENDICITIS(1) Catarrhal type.

This type is well illustrated in Specimen Nos. 10: 13: 22. Macroscopically, as shewn in the photographs, no gross pathological change, apart from increased vascularity of the subperitoneal vessels, is evident. The peritoneal coat is quite glossy; there is a slight thickening of the wall, and on section there is usually some slight swelling of the mucous membrane. But, on the whole, very little is to be seen on naked-eye examination.

Microscopically, catarrhal changes are found; these are sometimes limited to the mucous membrane, but frequently the other coats are also involved. The changes characteristic of this type are well shewn in examining Slide No. 10. Catarrh of the lining membrane and slight hyperplasia of the lymphoid nodes are noted in the upper half of the tube; the lower half shews an excessive increase of lymphocytic cells in the lymph nodes. Associated with this there is a polymorphonuclear leucocytic inflammation causing disorganisation of the various coats of tube with atrophy and thinning of the muscular and subperitoneal coats.

That a 'restitutio ad integrum' after catarrhal changes is not always the rule, because of the tendency for the deeper coats to be implicated, is borne out/

out by a study of Slide No. 7. Here, besides the usual degree of hyperplasia of lymphoid tissue, invasion of the muscular coat by fibrous tissue from the submucosa is seen. This would probably lead to subsequent stricture formation and would certainly predispose the organ to further infection. This is in accordance with the well-known aphorism -

"once appendicitis, always appendicitis."

(2) Ulcerative type

In this variety there is an extension of the pathological process with necrosis and desquamation of the mucosa. The characteristic changes are shewn in the photographs and sections of appendices, Nos. 6: 14: 20: 25.

Macroscopically, there is marked hyperaemia of the peritoneal coat, and the organ looks swollen and oedematous. In places, necrosis may be seen subjacent to the peritoneal coat and there may be a small perforation at this area. The association of ulceration with the presence of faecolith is shewn in Specimen No. 25; it is reasonable to suppose that this foreign body, either by increasing the virulence of the invading organism by preventing drainage from the lumen of the appendix to the caecum, or by causing pressure necrosis, would lead to further extension of the pathological process.

Microscopically, the characteristic changes are as/

as seen in Slide No. 20. Here the whole tube with its surrounding fat shews an intense inflammatory process with excessive fibrinous exudation rapidly becoming degenerated. The leucocytic and fibrinous exudate is causing extensive and general disorganisation of the structure of the organ. The lumen contains much necrosed fibrinous and cellular exudate which is adherent to the ulcerated mucosa. The epithelial lining is largely destroyed, and all trace of the lymphoid follicles in the submucosa is lost. The muscle fibres in the muscular coat are thin and separated by the leucocytic infiltration, while the sub-peritoneal coat is largely replaced by a thick layer of fibrinous and cellular exudate. All the tissues in the tube shew excessive invasion of leucocytes and œdema with softening and wasting. The surrounding fat tissue shews the same intense inflammatory process - congestion, leucocytic and fibrinous exudation.

While similar changes are to be found in the other specimens enumerated, the association of ulcerative changes are associated in No. 25 with a faecolith; in No. 14, with several colonies of bacteria - filamentous in character, possibly branching, and having a close resemblance to the colonies of Streptothrix actinomyces; and in No. 6, with an intestinal worm - Oxyuris vermicularis.

(3) Suppurative type.

Here, /

Here, the virulence of infection is such that liquefactive necrosis of the tissues is produced with resultant pus-formation. The naked-eye and microscopic appearances characteristic of this variety are to be seen in Specimens Nos. 8: 9: 17: 23: 27.

A special coloured drawing of Specimen No. 8 has been made for me by Mr. Richard Muir, and it illustrates the typical macroscopic appearances. Intense hyperaemia and oedema are present throughout all the coats, and small localised foci of suppuration are seen - chiefly in the submucous coat. The suppurative changes are most frequently found in the tip, but the whole organ may be converted into a pus-sac, the so-called empyema of the appendix. Perforation of the appendix is likely to occur in this variety, because of the excessive intra-luminal tension of the retained purulent contents.

Microscopically, the appearances are as seen in Specimen No. 9. The whole of the tube shews a diffuse inflammatory process, fibrinous and cellular in character. The lumen contains many inflammatory elements but little faecal matter. The mucous membrane shews small haemorrhages and leucocytic infiltration between the epithelial glands. In parts, the lining epithelium has been destroyed by suppuration. The submucous zone is markedly swollen by an acute suppurating process with destruction of the lymphoid tissue and surrounding connective tissue, and/

and in parts there is erosion through epithelial covering into the lumen. The suppuration is associated with hæmorrhage. The muscular coat shews leucocytic invasion and the muscle fibres are separated from each other by leucocytes and cedema. The same condition is seen in the connective tissue of the subperitoneal layer. There are no suppurating foci in either of these layers. The "pus area" is limited to the submucous layer. Adherent to the surface of the swollen and cedematous peritoneal coat is an irregular layer of fibrinous exudate - peritonitis. The peritoneal fat shews evidence of involvement in the inflammatory process and is covered with a thick layer of fibrinous exudate.

Slide No. 8 shews the characteristic disorganisation and degeneration of the lymphoid tissue and muscle fibres. Here, too, the mucous membrane - which is completely destroyed by the necrotic process - can be seen to fuse with the submucous, muscular, and subperitoneal coats. The association of local or spreading peritonitis with this type can thus be readily understood.

Slide No. 17 shews an acutely inflamed appendix generally, but the tip shews a greater disorganisation of tissue, and is converted into an abscess. The greater involvement of the tip is supposed to be due to its poorer blood supply; the meso-appendix with its contained artery, often stopping short of the tip./

tip.

No. 27 is interesting as it shews the association of suppuration with a fæcolith.

Suppuration within the appendix - as in any part of the body - would appear to be related to the following factors :-

- (1) Increased virulence of bacteria.
- (2) Weak resistance of tissues, e.g. following previous inflammation.
- (3) Obstruction of the lumen.
- (4) Presence of foreign body (fæcolith).

It must be stressed - as shewn in the slides above - that suppurative appendicitis is very liable to be associated with peritonitis, either

- or (a) local i.e. abscess.
- (b) spreading e.g. from sudden perforation of the organ.

(4) Gangrenous appendicitis

Death and putrefaction of the tissues 'en masse' are the outstanding features in this variety. The occurrence of such changes usually brings serious complications in its train, as there is no resisting barrier present to localise the infection to the appendix; contamination and involvement of the peritoneal cavity frequently but not invariably result. It is noteworthy that 81 cases of gangrenous appendicitis were found in the present series of 300 acute cases, i.e. 23 per cent. This would appear to be an exceptionally high percentage.

The whole range of changes characteristic of this type are illustrated by a study of Specimens 4: 5: 18: 24: 26.

Macroscopically/

Macroscopically, the appearances are well shewn by the photograph and coloured drawing of Specimen No. 4. The distal third of the organ is of a greyish green colour, and separated from the rest of the appendix by a zone of intense hyperaemia and inflammatory reaction. A circular zone of demarcation is readily visible between the two.

The distal portion is soft, disintegrated, offensive and very friable. The gangrene is always of the moist variety because bacteria are invariably present.

The gangrene may be localised to one small portion as in Specimen No. 18, where the tip alone is involved and forming a septic slough; or it may be widespread as in No. 5.

There may or may not be perforation, and in the present series, perforation occurred in almost 80 per cent. of gangrenous cases, viz., 62 out of 81 cases.

The site of perforation varies, but it more frequently involves the tip rather than the base. It seems to be frequently present over the projecting surface of a faecolith and examples of incipient perforation in the region of a faecolith are well shewn in Specimens Nos. 4 and 5.

Specimen No. 26 is of interest in that the gangrene was definitely limited to the mucous membrane. The microscopic changes are as shewn in Slide No. 4. Here, /

Here, extensive inflammatory and necrotic changes in the lower half of tube are associated with masses of retained faecal contents. The necrotic process has involved practically all the coats, mucous, submucous, muscular and peritoneal. At a point below one of the faecal concretions the wall is extremely thinned out so that perforation would soon have occurred. The acute inflammatory process is extending to the upper part of the tube especially along the sub-peritoneal coat. Vessels are distended and engorged with blood, and there is marked leucocytic infiltration of the connective tissue. A thin layer of fibrinous exude is present on the peritoneal surface. There is marked hyperplasia of the lymphoid tissue in the upper half of the tube and numerous lymphocytes have invaded the mucosa. The occurrence of gangrene may be attributed to several factors; for example, as in suppurative appendicitis it may result from :-

- (1) increased virulence of bacteria.— Streptococci have been associated with the most advanced pathological changes in the appendix and Williams' work suggests that the bacillus of gasgangrene may be an important contributing agent.
- (2) weak resistance of tissues, e.g., following previous inflammation.
- (3) obstruction of the lumen. Wilkie of Edin. has made an important contribution in suggesting that there are two types in appendicitis (a) Acute inflammatory appendicitis and (b) Acute obstructive appendicitis. It is certainly a fact that gangrene is much more common in the latter type. Obstruction of the lumen, then, would seem to predispose to the development of gangrene. (Behan's experiments).

(4)/

- (4) presence of a foreign body.— A faecolith, either by inducing pressure necrosis or by exalting the virulence of the organism or by obstructing the lumen, may give rise to gangrene. But this is not invariably so.

PERFORATION OF THE APPENDIX

Rupture of the appendix is the dreaded complication in appendicitis as peritonitis with its concomitant evils supervenes. Patients die, not from appendicitis as such, but from its complications -- the chief and most dreaded of these being peritonitis. It is, therefore, important to consider the factors concerned in the production of perforation. It has already been shewn that this may occur in ulcerative type, suppurative type and in the gangrenous type (especially the last). Perforation would seem to be associated with the following :-

- (1) Concretion: This causes pressure necrosis and perhaps obstruction. Attempts of the organ to expel the concretion may result in perforation at the site of necrosis. Specimen No. 27 shews a perforation about to occur over the projection surface of the concretion. Many observers state that concretions are the chief cause of perforation, but I have had many cases of perforated gangrenous appendices where no concretion was found.
- (2) Obstruction of the appendix: Wilkie regards acute obstructive appendicitis as being a form of acute intestinal obstruction with the same liability to gangrene and perforation. Obstruction may be due to excessive faecal content; to swelling of the mucous membrane; to presence of concretion; to kinks and angulations; or to presence of carcinoma of, or stricture of, the appendix.

(3)/

- (3) Diet: It has been shewn by experimental investigation in animals (Wilkie) that more advanced pathological changes are associated with protein diet. This may be a factor in the production of gangrene or perforation.
- (4) Personally, I am convinced that the commonest cause of perforation is the administration of a purgative. This, as already shewn, cuts off the blood supply and causes excessive muscular spasm. If the appendix be obstructed from any of the above-mentioned causes, the sudden increased peristalsis leads to further increase of intra-luminal tension which can only be relieved by perforation. The effect of giving a purgative in such circumstances would appear to be analogous to the giving of pituitrin in cases of obstructed labour with the same disastrous result, viz., rupture of the organ (uterus).

B. CHRONIC APPENDICITIS.

This is a problem unto itself. Difference of opinion exists as to whether the condition arises de novo, or if it is merely the result of a previous acute attack. Again, there is lack of unanimity as to the pathological changes characteristic of the condition, and there can be ^{no} doubt that "chronic appendicitis" is too frequently diagnosed.

Specimen Nos. 1, 2, 3, 11, 15, 16 and 21 are true examples of chronic appendicitis, and shew the various noteworthy pathological changes.

Macroscopically, as in No. 1, the tube may be acutely kinked, while - as in No. 2 - a stricture of the lumen may be present. Complete obliteration of the/

the lumen is rare, but such a type is seen in No. 21.

The characteristic findings are a thickened, avascular organ, very often bound down by adhesions to other organs or to the peritoneum of the right iliac fossa: the thickening chiefly involves the submucous coat.

Microscopically, the changes are as seen in Specimen No. 15. Here, the mucosa shews little change; the submucous coat is fibrosed, the lymphoid tissue atrophied and the muscular coat hypertrophied. The subperitoneal coat is thickened and fibrous with lymphocytic cells infiltrating the fibrous tissue. Along the perivascular lymphatics near the surface, numerous small haemorrhages have occurred. The fibrous stroma in the fat surrounding the tube is thickened, and small masses of fibrin are adhering to the surface of the fat lobules.

Specimen No. 21 is interesting. Here, the whole length of the tube lumen appears to have been completely obliterated. There is no evidence of mucosa or lymphoid tissue, these being replaced by well-formed fibrous tissue. At points along the central line in this fibrous tissue are small cellular areas composed of lymphocytes, and between the collagen fibres are numerous vacuolated mononucleated cells "foamy" cells or phagocytes. The muscular coat shews much interstitial connective tissue between the muscle fibres. This suggests a condition of chronic/

chronic replacement fibrosis with obliteration.

It is noteworthy that different views are held even as to the nature of this obliteration. Ribbert submitted that the changes were involutionary; while Berry and Lack (already quoted) state that the condition is pathological and not physiological in origin.

II.

COMPLICATIONS.

(Spread of Disease).

The spread of disease from the appendix, as from any other organ may occur in 3 ways :-

- (a) By contiguity of tissue.
- (b) By blood stream.
- (c) By lymphatic stream.

(a) Contiguity of tissue

As we have already seen whilst studying anatomical relations, the appendix may be entirely retro-peritoneal, lying in the cellular tissue of the posterior abdominal wall. In such cases, appendicitis may lead by contiguity of tissue to retro-peritoneal cellulitis, a condition difficult to localise and of serious import. Again, the contiguous caecum may be involved - caecal fistula resulting; while in the female, disease may spread via the ligament of Clado or genito-mesenteric fold to the uterine appendages and explains the frequent association of appendicitis and appendagitis.

By/

By far the most important tissue to be involved from extension of disease from the appendix is the peritoneum, and it cannot be too strongly emphasised that the commonest cause of peritonitis is to be found in inflammatory lesions of the appendix. This fact makes this insignificant looking organ rank high in surgical importance. As patients die, not from appendicitis as such, but from its complications, the chief and most dreaded of which is peritonitis, it behoves us to study very briefly the function and pathology of the peritoneum.

Peritoneum:

The peritoneum has two coverings - an outer parietal layer, and a visceral layer which forms a covering to the abdominal viscera. The visceral peritoneum is insensitive but the parietal layer is extremely sensitive. John Morley of Manchester regards this layer as being a great protective mechanism to the body.

There are two specialised portions of peritoneum - one which forms a support to the viscera and is designated mesentery and another which is capable of mobility called the omentum.

In structure, the peritoneum is a serous membrane composed of an endo-thelial layer and a basement membrane, and is richly supplied with lymphatics and blood vessels. It has two chief functions - that of absorption/

absorption including phagocytosis, and exudation. It has been shewn by experiments that particles of carbon and dyes when injected into the peritoneal cavity are readily phagocyted by mononuclear cells and absorbed; while its exudative powers are seen in the ascitic form of tuberculous abdomen or after perforation of gastric ulcer. Deaver states that "absorption depends upon the pressure of the abdominal muscles and the respiratory contraction in which the diaphragm plays a most important rôle, for by its contraction and relaxation, fluids and particles are aspirated from the peritoneum and forced onward. The size of the peritoneum or its surface area is equal to that of the skin covering of the body - 17,500 sq. ins."

It would appear that the different areas of the peritoneum possess different rates of absorption, the order of activity being diaphragmatic, omental, and enteronic.

Exfoliation of the endo-thelial layer may produce granulations and form adhesions which may be either defensive, e.g., walling off an abscess, or offensive, e.g., leading to intestinal obstruction.

Exudation would appear to be entirely protective and defensive in nature. The exudate with fibrinous deposits engulfs micro-organisms which are later phagocyted. As Deaver says "The insulted peritoneum weeps, and by its tears it defends itself."

The/

The mesenteries act as suspensory ligaments to the viscera and transmit blood vessels and nerves to them.

The omentum has the important asset of mobility. It migrates to seats of inflammation. Rutherford Morrison of Newcastle terms it "Policeman of the abdomen."

Other observers, because of its ability to seal over a perforation of the stomach, duodenum, or appendix, refer to it as "the abdominal plumber;" and Moynihan calls it "the great drain pipe."

Pathology of peritonitis:

The types of inflammation like that of any serous membrane may be of the following :-

- (1) Serous.
- (2) Sero-fibrinous.
- (3) Sero-purulent.
- (4) Purulent.

The question as to whether the peritonitis will be localised or of the spreading type will depend upon the resistance of the tissues; the virulence of the infecting organism; and the time interval between the onset of the disease and the involvement of the peritoneum. Sufficient time is necessary for the mobilisation of the mesentery, the exudation of anti-bactericidal fluid and the formation of adhesions to localise or neutralise the infection. If insufficient time be given, no defence is possible and the peritoneum is flooded with highly toxic material which/

which soon passes into the general system. The portion of the peritoneum involved will depend upon the position of the appendix; thus, the peritoneum behind and around the caecum is most frequently involved while the pelvic peritoneum is next in order of frequency. The evil effects of peritonitis are largely due to the inherent physiological property of the peritoneum for absorption. Toxins are absorbed either through the blood or lymphatics and produce marked toxæmia. Costain believed that, by draining the thoracic duct the amount of toxic absorption could be greatly diminished, but Paterson Brown of Edinburgh has brought forward experimental evidence to negative this suggestion.

Again, the fact that the intestines are being bathed in toxic peritoneal fluid may lead to paralytic ileus or the form of "ileus duplex" of Sampson Handley seen in cases of pelvic peritonitis. Williams' work suggests that the paresis of the bowel due to the toxic action of septic peritoneal fluid allows of absorption of bacterial poisons produced within the lumen of the bowel. The organism concerned is usually the bacillus of gasgangrene and he recommends the administration of anti-gasgangrene serum in such cases.

The mesentery of the appendix may become gangrenous and lead to suppurative pylephlebitis via the ileo-colic vein, and we have already seen that some operators/

operators ligate the vein in all cases of gangrenous appendix to prevent liver abscess.

It has been stated above that the omentum is a great defensive mechanism but Wilkie considers it to be responsible for spreading the disease to the upper abdomen especially the stomach and duodenum via retrograde omental venous emboli. He says: "The extraordinary faculty possessed by the omentum for plastic adhesion to any zone of peritoneal irritation, and thus for walling off and localizing inflammatory processes, is so constantly evident to the surgeon and to the pathologist, that they have almost come to regard the great omentum as a purely beneficent agent in abdominal pathology. Notwithstanding its peculiar capacity for rapid reaction and repair, it would, indeed, be strange if the omentum could take part in so much inflammatory trouble and yet come out scathless. That a chronic inflammation of the omentum may remain after the primary seat of infection has healed has been repeatedly demonstrated, but that the omentum may form the channel by which the pathological process may be carried to other organs is a fact that has hitherto received but scant recognition. The intimate anatomical relations of the omentum with the stomach and the first part of the duodenum, especially in regard to their vascular supply, suggest the possibility of morbid conditions in the former spreading or being conveyed to the latter through their vascular/

vascular connections." He then proceeds to show how gastric and duodenal ulcer and gastric erosions may result from retrograde venous emboli originating in thrombosed omental veins.

Thus, it will be seen that the normal physiological properties of the peritoneum, mesentery and omentum may be the means of extending to other parts of the body pathological processes which originate in the appendix.

This brief study of the pathology of peritonitis suggests the following régime to limit toxic effects and absorption.

- (1) Removal of toxins by drainage at operation.
- (2) Lessening the absorption.
 - (a) by giving morphia which lessens peristaltic action of the bowel, diminishes the excursion of the diaphragm, and reduces intra-abdominal pressure by relaxing the abdominal muscles.
 - (b) by ligating the ileo-colic vein in gangrenous appendicitis and by stimulating the function of the liver to form antibodies by applying hot fomentations over the right hypochondrium.
 - (c) by giving anti-gas gangrene serum.
- (3) By Posture.

Fowler accidentally discovered that patients in a sitting posture after an abdominal operation progressed more favourably than those treated in a recumbent posture. This is explained on the basis of slower rate of absorption of the pelvic peritoneum over the diaphragmatic peritoneum. Fowler substantiated this position - now called the Fowler position - on two/

two assumptions (1) that the absorption from the peritoneum takes place chiefly from the diaphragmatic surface and (2) that it takes place directly into the lymphatic system. Dandy and Rowntree criticise this assumption and state that peritoneal absorption is practically equal in all postures except when the pelvis is down, in which case it is 15 per cent. less than in the others. They state that gravity has a decided effect and that the Fowler position is not justified as supposed but because of the 15 per cent. lessened absorption by the pelvic peritoneum.

Whatever be the rationale, there can be no doubt that the introduction of the Fowler position is one of the most important advances in surgery and has led to an extraordinary diminution of mortality rate and of the incidence of sub-phrenic abscess.

We have briefly considered the practical suggestions to be carried out to prevent toxic absorption once peritonitis has arisen, but greater emphasis will be given later as to how to prevent peritonitis ever arising at all in cases of appendicitis.

(b) Spread by blood stream.

Reference has already been made to spread via the ileo-colic vein, and the thrombosed omental veins. Mention must also be made of the occurrence of phlebitis of the femoral vein and its tributaries usually on the left side. How this arises is not entirely clear; it seems to be common in puerperal sepsis, and 6 out of my series of 600 cases of appendicitis had this complication. Four occurred in women, three of/

of whom had thrombosis of the femoral vein and one thrombosis of the calf; two occurred in men, and in both the calf veins were involved.

(c) Spread by lymphatic stream.

It is very common in cases of appendicitis and especially in children to find enlargement of the ileo caecal glands. It is difficult to be sure in such cases whether the enlargement follows infection of the appendix or if it is tuberculous in origin. Costain's assumption of spread via the thoracic duct into the circulation has already been referred to and the very interesting work of Braithwaite on the flow of lymph from the ileo-caecal angle has been quoted in the section on Anatomy. He regards duodenal ulcer, gastric ulcer, and cholecystitis to be due to retrograde aberrant lymphatic flow from the appendix.

Foster, writing on the association of pneumonia with fulminating appendicitis says, "The late J. B. Murphy well proved the fact that lymph current proceeded from the root of the caecum up the ascending colon to the diaphragm. On the abdominal surface of the diaphragm the lymphatic distribution is especially multiple, with a relatively high pressure from the ascending colon upward. This minute lymph capillary distribution is also present in the thoracic surface of the diaphragm. Here, however, we have a relatively lower intro-channel lymph pressure because of the smaller volume of lymph above the diaphragm as compared/

compared with the amount below the muscular dome. Thus it will be seen that, with a complete anastomosis between the lymph capillaries of the two diaphragmatic surfaces, the pressure from below upward will tend toward metastasis in that direction. Again, Murphy has proved that such metastasis in this locality takes place through the lymph rather than other circulatory channels. Admitting this to be a proved fact coming from such a high authority, we should respect the lymphatic circulation the more and ever regard its activity."

In this connection, Kelly states that "in every case of empyema in children, particularly in right sides affections, and above all when the pus is ill-smelling, the surgeon should bear in mind the possibility of this being a complication of appendicitis and that he should make such examination of the right iliac fossa (rectal above all !) as shall decide the question."

Appendix as a focus of sepsis:

It has been suggested that the appendix is a focus of sepsis whence bacteria may be distributed to other parts of the body. Moynihan, Paterson, Wilkie and others regard cholecystitis, gastric ulcer, and duodenal ulcer to be frequently secondary to a diseased appendix- "The infective agent which reaches the gall-bladder in this way is derived from the portal system. In this are two streams - the one derived/

derived from the alimentary canal, the other from the spleen. So far as the alimentary stream is concerned the main source of origin (of any infection) is most frequently the appendix. We know, from the clinical experience of a multitude of observers, that the appendix shews evidence of disease in a large proportion of cases in which cholecystitis or gastric or duodenal ulcers are present. Indeed, it is rare to find solitary inflammatory affections of the stomach, duodenum, pancreas, liver, gall-bladder or appendix. When one of these shews evidence of disease, one or more of the others is likely also to be implicated." (Moynihan).

And again, Heyd states, "The normal post-mortem rate of diseased appendices is approximately 17 per cent. and it is no accidental finding to observe that in disease of the gall-bladder a partially, or completely obliterated appendix is found in 55 per cent. of the cases and, in gastro-duodenal ulceration, in 60 per cent. of the cases. These facts would suggest the possibility of a septic leakage by way of the portal system with infection of the periportal veins and then a hepatic lymphangitis with a secondary infection of the gall-bladder, representing in effect a sequential infection of the gall-bladder through the lymphatic system."

Mitchell also refers to cases of migraine as being cured by appendicectomy and I have notes of two cases/

cases of appendicular abscess, where the abscess was merely drained at first, which were followed by septic arthritis of the spine - which condition was relieved by appendicectomy. However, a caveat should be given against regarding the appendix as the primary cause of all chronic maladies as appendicectomy must not be considered "a panacea for all ills."

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CHAPTER V.

S Y M P T O M A T O L O G Y

We now proceed to analyse the symptoms of the condition, to evaluate their significance in diagnosis, and to point out the fallacies in their interpretation. In this and subsequent sections we are concerned chiefly with acute appendicitis, the problems of chronic appendicitis being dealt with in a separate chapter. We shall consider (I) the general symptomatology and (II) additional features due to the various positions occupied by the appendix.

(I) General Symptomatology

The symptoms of acute appendicitis may be either (A) inflammatory or (B) obstructive in origin.

(A) Inflammatory symptoms are due to toxic absorption and include pain; tenderness, superficial and deep; rigidity; leucocytosis; temperature; and pulse.

(B) Mechanical or obstructive symptoms result from interference with the normal peristalsis and include colic; nausea or vomiting; constipation or diarrhoea.

(A) INFLAMMATORY:

Pain. As this is the earliest, the most constant and the most reliable symptom, it is necessary to consider it at some length; and first of all it will be useful to recall very briefly the nerve supply of the abdomen and the pathway of sensation from the abdomen to the sensorium in the brain. The skin of the/

the anterior abdominal wall is supplied by the lower six thoracic nerves and the first lumbar. The ileo-hypogastric and ileo-inguinal branches of the lumbar nerve supply the hypogastric region, the skin over the pubes, the inner aspect of the thigh, and the upper part of the scrotum. The tenth thoracic supplies the skin in the region of the umbilicus. These nerves run obliquely round the chest. The eighth intercostal nerve is placed posteriorly at the inferior angle of the scapula; anteriorly it terminates midway between the xiphoid process and the umbilicus. The skin of the posterior abdominal wall is supplied by the posterior division of the same nerves. The anterior divisions of the first and second lumbar nerves, with the lower thoracic nerves, are connected with the corresponding ganglia of the sympathetic, with both white and grey rami communicantes.

The nerve supply of the abdominal viscera is derived from the lower sixth thoracic and the first lumbar segments, and the afferent impulses from the viscera reach the same segments of the spinal medulla, as do the afferent impulses from the skin of the abdominal wall. (Huey).

Fraser of Edinburgh in a masterly survey of "Pain in the Iliac Fossa" — to which further mention will be made later — describes the pathway of sensation from the abdomen thus : "The stimulus (which produces/

produces pain) has travelled along a well-defined route; it has been transmitted by a mixed nerve to the spinal cord, it has entered that organ through the medium of the posterior root, and has terminated for the time being in the synapse of the grey matter of the posterior horn. Picked up at this point by the synapse of the second axone, it passes to the opposite side of the cord, and then, by way of the spino-thalamic tract and the fillet, it ends as a synapse in the outer part of the optic thalamus. A third and last axone proceeds from this point in the posterior part of the internal capsule to end in the sensory area of the cortex which lies behind the fissure of Rolando. It is there that the actual consciousness of the peripheral stimulus is recorded."

Edwin Bramwell states that: "The nerve impulses in the optic thalamus are appreciated in terms of pain and are recognised as diffuse, unlocalizable, disagreeable sensations. The function of the cerebral cortex, which is directly connected with the thalamus by fibres arising in the latter, is to localize these sensations and to analyse their significance."

The recognition that the fibres for pain are in close association with the fibres for heat in the cord is the physiological explanation of the relief given by counter irritation to painful areas.

The mechanism and significance of pain as a symptom of disease has formed the subject of many contributions/

contributions to medical literature. Hilton's "Rest and Pain", the late Sir James Mackenzie's "Interpretation of Symptoms" and the work of Sir Henry Head have long been regarded as classical; while more recently the contributions of Hurst, Poulton, Fraser, Wilkie, Morley, Ryle, Bramwell, Mayo and Buzzard have resuscitated interest in the subject. The question as to whether the viscera are sensitive to pain, and the views as to the explanation of associating tenderness and rigidity in visceral inflammations have been keenly debated. The evolution of our knowledge of abdominal pain has been summarised thus by John Morley of Manchester: "Almost the first considerable contribution to our knowledge of the mechanism of abdominal pain was made by J. Ross of Manchester in 1887. Ross held that there were two kinds of pain in visceral disease - true splanchnic pain, felt in the affected organ; and associated somatic pain, referred to the cerebro-spinal nerves of the body wall. Lennander established the insensibility of the exposed gastro-intestinal tract to the ordinary painful stimuli, and attributed all abdominal pain to stimulation of nerves in the sensitive parietal peritoneum. Mackenzie, whose views form the orthodox teaching of the current text-books, accepted Ross's views on somatic pain, but rejected splanchnic pain as non-existent. He believed that painful stimuli passed from the viscera through the afferent splanchnic/

splanchnic nerves to the spinal cord, but were only appreciated by the brain as arising from the sensory nerves of the parietes. Hurst in 1911 published observations which have restored our belief in splanchnic pain; he proved that this type of pain is produced by a single adequate stimulus - namely, increased tension in the muscular wall of the viscus concerned."

Mackenzie in his book states that: "Visceral pain is almost certainly a referred pain; the pain is not felt in the organ itself, but in the area of distribution of those spinal nerves whose nuclei are in close connection with the spinal nuclei of the afferent sympathetic fibres from that organ. The fact that these spinal nerves frequently supply the skin and muscles over the particular viscus with which they are indirectly associated gives rise to erroneous impression that the pain and tenderness are in the viscus itself."

Morley disagrees with Mackenzie. He shews that true splanchnic pain does exist and that deep tenderness and muscular rigidity result, not from the viscerocutaneous and visceromuscular reflexes (Mackenzie) but rather from peritoneocutaneous and peritoneomuscular reflexes. According to Morley, there are two types of pain, each with a different explanation (1) the initial or splanchnic pain and (2) a secondary pain due to involvement of the parietal peritoneum.

Initial/

(1) Initial pain: This pain frequently begins in the early hours of the morning or after dietetic indiscretions. It is felt in the centre of the abdomen passing across the umbilicus or epigastrium and is very imperfectly localised, Its onset is sudden and in those apparently enjoying good health. The patient thinks he has indigestion, colic or wind, such is the location of the pain. Sherren of the London Hospital states that: "In a first attack of acute appendicitis the pain never starts in the right side. Appendicitis should be the last, not the first, disease to think of in a patient with pain starting in the right side of the abdomen."

Fraser agrees with Sherren and states that "if pain begins and remains in the right iliac fossa, the chances are that the case is not one of appendicitis."

The paroxysmal or colicky nature of the initial pain cannot be too strongly emphasized and is sometimes referred to as appendicular colic. It is not associated with any tenderness on palpation and the patient may try to secure relief by pressing on the abdomen -- a thing, as Morley says, he never attempts to do when the second pain has appeared.

Morley states that "the initial pain is a true splanchnic pain and is felt in the umbilical or lower epigastric region and not in the region of the appendix. The appendix is developmentally a part of the mid-gut/

mid-gut. The brain can only appreciate painful stimuli arising from any portion of the mid-gut as vaguely situated in the centre of the abdomen."

As to the cause of this initial pain most observers are agreed. It is due to increased tension in the muscular wall of the viscus concerned. (Morley)

"The nature of the stimulus which induces visceral pain is still a matter of controversy, but it is evident that hollow viscera are particularly sensitive to stimuli which arise in association with undue contraction or undue relaxation of the muscular walls." (Fraser).

"It is a basal fact in clinical observation that most abdominal pains, if we exclude those due to irritation of the parietal peritoneum, are due to spasm of, or tension in, the hollow viscera." (Wilkie)

Ryle concludes that "visceral pain is due to an abnormal increase in tension in the muscular element of the wall of the viscus" and he has drawn up "Laws of Visceral Pain" some of which we quote.

(1) "Visceral pain has its origin in, and is due to an abnormal increase in tension of the muscular element of the wall of the viscus, this increase in tension resulting either (a) from contraction of the muscle, or (b) from its failure to relax in face of increasing intravisceral pressure."

(2) "Relieving factors in visceral pain, and other than those which merely deaden consciousness, are/

are invariably factors which reduce intravisceral pressure or encourage muscular relaxation, e.g., the relief of pain from the sudden perforation of a diseased appendix."

(3) "As would be anticipated if the truth of (1) and (2) is conceded, the severity of mechanically induced pain is in inverse proportion to the normal distensibility of the viscus. (Thus the most severe pains are found in disease involving tubes of small calibre and small distensibility, such as the ureter, the bile ducts, and the arteries; the more bearable pains, in disease involving organs of wide calibre and a wide range of physiological distensibility and postural adaptibility, such as the stomach and urinary bladder.)"

(4) "Visceral pain, having its origin in muscle, is related to the fundamental activity of the affected viscus."

This initial pain usually lasts from 12 to 36 hours but before the cessation it has usually given place to the secondary pain.

(2) Secondary pain: The second or localised pain usually comes on when the initial splanchnic pain is diminishing in intensity. It is due to the involvement of the neighbouring parietal peritoneum; its localisation, therefore, depends almost entirely upon the position of the appendix. It is usually in the right iliac fossa or may be in the left side of the/

the abdomen or pelvis if the appendix overhangs the brim of the pelvis. In the retro-caecal appendix the pain may be localised to the right renal angle or to the region of the gall-bladder. The secondary pain has also a different character and is accentuated by any movement of the abdominal muscles such as occurs in coughing, vomiting, deep breathing, or adoption of sitting-up posture. Respiratory excursions of the lower abdominal wall are limited, the right iliac fossa becomes tender on palpation, the patient remains perfectly quiet in bed and, in contrast to the initial pain, resents any pressure or palpation. The inflammation of the parietal peritoneum may follow bacterial invasion or chemical irritation. Morley states that it is only when the parietal peritoneum becomes involved that hyperalgesia, deep tenderness and muscular rigidity result. This he explains on the basis of a peritoneo-cutaneous and a peritoneo-muscular reflex. Fraser states that there are three pains; the first, due to the severe spasmodic contraction of the related hollow viscera, the lowest coils of the ileum, the cæcum, and the pyloric portion of the stomach, is located to the epigastrium or umbilicus; the second, due to increased tension within the appendix, is transmitted through the medium of the tenth, and eleventh dorsal nerves to the subumbilical region on one side of the mid-line; the third, due to an infection of the related/

related peritoneum delimits itself at the situation which the appendix occupies.

Both Morley and Fraser are agreed that the pain in appendicitis does not usually begin in the right iliac fossa. "Right iliac fossa pain as an evidence of appendicitis", says Fraser, "depends upon the organ occupying what is supposed to be its normal anatomical position and, moreover, its appearance is an evidence of a stage in the local pathology which bespeaks an established infection of the organ with peritoneal involvement."

It will be seen from the above that the visceral and parietal peritoneum are affected by different stimuli. The initial pain of appendicitis is visceral in origin, is situated in the umbilicus and is accompanied by tenderness or rigidity. The secondary pain is due to involvement of the parietal peritoneum, is usually localised to the right iliac fossa but may vary according to the position of the appendix. The pain in this type is usually accompanied by deep tenderness and muscular rigidity.

The following clinical history seems to support Morley's assertion.

C.D.J. - female - aged 18: was admitted to hospital as an emergency. Thirty-six hours before admission, she complained of pain shooting across the umbilicus. One hour later she vomited twice, and twelve hours after the onset of the initial symptoms, pain became localised to the right iliac fossa.

Eighteen hours ago she took two castor oil pills./

pills. Two hours after admission, she complained of violent umbilical pain and this was followed half an hour later by a more diffuse pain in the right iliac fossa.

The exacerbation of umbilical pain would appear to be due to peristaltic efforts of the appendix to rid itself of faecal material, etc. consequent upon purgation. This being a visceral pain, was referred to the umbilicus. The appendix had perforated at operation and no doubt the increase in extent of pain in the right iliac fossa was due to the greater involvement of the parietal peritoneum consequent upon perforation.

As Bramwell states, "Pain is indeed a protective danger signal and since it affords a warning that all is not well, it fulfils an invaluable purpose. In so far as pain is informative it must be regarded as an indispensable blessing, while it is at the same time one of the curses to which human flesh is heir."

Weiss and Davis have been able, by injecting novocain locally, to cause disappearance of the pain and Morley adduces from their work confirmation of his theories of the peritoneal reflexes to skin and muscle. Pain may also be relieved by restriction of food and the Fowler position; and may be aggravated by coughing, taking of food and, most important of all, by taking an aperient.

There are certain pitfalls in the interpretation of pain as a symptom of appendicitis :-

- (1) Pain does not begin in the right iliac fossa.
- (2)/

- (2) Pain may never be in the right iliac fossa if the appendix is retro-caecal or in the pelvis, or in the left iliac fossa if the appendix is in the left iliac fossa due to mal-rotation of the gut.(N.M. Dott).
- (3) There is no accurate method of assessing the amount of pain and this entirely personal factor must be assessed by the clinical acumen of the surgeon.

Tenderness : This symptom is allied to that of pain and is the next most valuable symptom. It is present after the first, or initial splanchnic pain, has ceased, and is present so long as the parietal peritoneum is inflamed. Deep tenderness is practically never absent in cases of acute appendicitis. Tenderness may be of two types:-

- (1) Superficial - Hyperæsthesia or hyperalgesia.
- and (2) Deep tenderness, i.e., tenderness on deep palpation.

(1) Hyperalgesia: This sign is not sufficiently looked for. It may be tested for by stroking the skin with a pin, or by plucking the skin away from the deeper structures. It is customary to begin in the left iliac fossa and work round to the right iliac fossa. If the patient experiences increase of pain in the right iliac fossa during the above, hyperalgesia is said to be present.

Different estimates are given as to the diagnostic significance of this symptom. Sherren finds it present in 30 per cent. of cases and describes an area of hyperæsthesia to be found. This triangle of Sherren/

Sherren is formed by joining the umbilicus, the right anterior superior iliac spine, and the pubic spine. He makes the following conclusions from his observations.

1. Cutaneous hyperalgesia is probably present at some time during all first attacks of appendicitis except perhaps in the fulminating type and depends upon tension within the appendix.
2. It may be absent in attacks after the first, if the first was of sufficient severity to destroy the nerve tissue in the wall of the appendix.
3. When present in attacks subsequent to the first, it often persists long after all other signs of the disease have gone owing to the tension within the appendix being kept up by the pressure of a stricture.
4. It gradually disappears during convalescence as the other signs of the disease clear up.
5. Disappearance of cutaneous hyperalgesia without improvement in the general condition of the patient is a sign of perforation or gangrene of the appendix and should be a signal for immediate operation.
6. The presence of cutaneous hyperalgesia is no contra-indication to operation. Abscesses may form and general peritonitis may develop while it is present.
7. Its absence, on the other hand, is of great importance. Absence of cutaneous hyperalgesia, the patient coming under observation early in the first attack of appendicitis, is a sign of gangrene of the appendix unless the case is obviously a mild one and the patient is rapidly getting well.
8. Cutaneous hyperalgesia is as a rule absent in cases of abscess of the appendix.
9. The age of the patient and the position of the appendix have no influence upon the cutaneous hyperalgesia.
10. It is occasionally of use as an aid to diagnosis of appendicitis.

Mansell Mouillin regards hyperæsthesia, when associated with other evidence pointing to inflammation of the appendix, to be a clear indication that the wall of the appendix itself is involved and that, therefore, though the inflammation subside, it will in all probability leave some permanent alteration in the appendix which will necessitate operation later. He stresses the importance of sudden cessation of hyperaesthesia. Such cessation, without at the same time any corresponding improvement in the general symptoms, suggests very strongly that the appendix has become gangrenous and that immediate operation is necessary to prevent septic peritonitis.

Morley, however, is more sceptical of the value of hyperalgesia. "A phenomenon that can be detected by different observers with a frequency that varies from 21 per cent. to 59 per cent. would appear to be of but little help in diagnosis. Unless, given certain pathological conditions in the appendix, hyperalgesia can be detected as a constant phenomenon, I hold that it is merely of academic interest. A further consideration which invalidates hyperalgesia as an aid to diagnosis is the extreme frequency with which it can be elicited over the appendix in the neurotic abdomen, or those cases simulating chronic appendicitis, where on exploration no evidence of organic disease in the appendix or any adjacent organ can be found."

(2) Deep tenderness: This is elicited by palpation with the flat of the hand beginning in a non-painful area and working round to the right iliac fossa, the facial aspect of the patient being observed to see if pain is experienced. The point of maximum intensity of tenderness was first accurately described by McBurney in 1889 and may be defined as a point at the junction of the outer and middle thirds of a line drawn from the right anterior superior iliac spine to the umbilicus and is referred to as 'McBurney's Point'. The site of maximum intensity varies, however, according to the position of the appendix and, while McBurney's point will be diagnostic when the appendix is in the right iliac fossa, other points have been described for other positions of the appendix, e.g., (1) Monroe's point - which lies at the intersection of the McBurney line with the outer border of the right rectus - when the appendix is pointing towards the spleen. (2) Morris' point, which is one and a half inches from the umbilicus on the same line and (3) Lanz' point which is situated at the junction of the right and middle third of a line between the two anterior superior iliac spines when the appendix is in the pelvic position.

It must be remembered that tenderness may be elicited only on rectal and vaginal examination and no case of appendicitis should be diagnosed or operated upon in which either or both of these examinations/

examinations have not been made.

The contrast as to the methods of diagnosis has been laconically described thus:-

The Surgeon	diagnoses	p.r.	(per rectum)
" Gynæcologist	"	p.v.	(per vaginam)
" Physician	"	p.m.	(post-mortem)

Whether or not this be true, it is certain that many cases of pelvic appendicitis have been missed because of failure to do a vaginal or rectal examination.

It will be seen that deep tenderness is a very reliable sign in appendicitis and possesses a significance of such great value that "the surgeon" as Kelly says "hesitates to make a diagnosis in its absence."

The following pitfalls should be remembered in its interpretation -

- (1) Abdominal tenderness may be absent in
 - (a) retro-caecal appendicitis and
 - (b) pelvic appendicitis.
- (2) When palpating in the left iliac fossa, pain may be experienced in the right iliac fossa. This is called Rovsing's sign - is present in appendicitis and is due to gas being forced from the pelvic colon to distend the caecum.
- (3) Two areas of tenderness on palpation may be found, e.g., in the epigastrium and in the right iliac fossa, and it may be difficult to know if appendicitis is present. Dowden's (Edinburgh) method clears up this difficulty. One hand is kept on the epigastric area, and the other passed over the right iliac fossa. If tenderness now disappears in the epigastrium, appendicitis is the likely diagnosis.

Rigidity.

This/

This is next in importance as a diagnostic symptom or sign. It depends for its presence on the peritoneo-muscular reflex of Morley, and denotes involvement of the parietal peritoneum.

It is usually localised from the first, as rigidity does not accompany initial or splanchnic pain. It is exactly delimited according to the position of the appendix, and hence is most common over the right iliac fossa.

When the appendix has perforated, the area of rigidity will extend pari passu with the area of peritoneal involvement.

In this case it may occasionally be difficult to distinguish this from general voluntary rigidity, but examination under anaesthesia will usually differentiate. Rigidity is a useful sign but varies in position according to the position of the appendix and hence the following fallacies are to be guarded against :-

(1) Abdominal rigidity may be absent in

and (a) retro-caecal
 (b) pelvic appendicitis.

(2) In appendix abscess with omentum lying between the appendix and the parietal peritoneum, rigidity may be slight, or even absent.

Pain, Tenderness and Rigidity form the three cardinal symptoms in appendicitis. As the other symptoms are of little diagnostic significance, they need not detain us long.

Leucocytosis/

Leucocytosis.

This is merely a symptom of the disease and is only of value when considered with the other symptoms, otherwise it is not only worthless but may be misleading. Hewitt says that "the absolute count when taken alone is in the great majority of cases a reliable index in diagnosis. The correlated absolute and polynuclear counts are of greater value especially as regards prognosis. A high absolute count with a high polynuclear count means a good prognosis (e.g. absolute 35,000 polynuclear 95 per cent.) A low absolute count with a high polynuclear count (e.g. 7,000 with polynuclear 95 per cent.) indicates a grave prognosis.

Temperature and Pulse.

The important point is that severe forms of appendicitis may be present without alteration of the temperature or pulse. An increase of temperature will only be present when toxins are absorbed either in the suppurative type, or perhaps only after peritonitis has supervened. The pulse rate is usually more reliable but Wilkie issues a warning in cases of acute obstructive appendicitis. He says, "To recognise the obstructive cases in their early stages, the state of the temperature and pulse must be ignored and diagnosis based entirely on the facies and the local examination of the abdomen."

B. MECHANICAL or OBSTRUCTIVE SYMPTOMS:

Colic/

Colic.

This has already been dealt with under splanchnic pain.

Nausea or Vomiting.

This always succeeds the initial pain - a few hours intervening. It is my experience that the patient only vomits three times - never more than five times. If vomiting persists, other causes such as renal, colic or biliary colic are to be suspected or spreading peritonitis has been superimposed upon appendicitis. Lockwood explains the symptoms as due to involvement of the solar plexus while Morley regards it as being due to a splanchnic inhibitory reflex originating in the parietal peritoneum and is provided as an indication of the danger of intake of food. The late Alexis Thomson used to say that the stomach was the spokesman of the alimentary tract and "in lesions of the abdomen what could the stomach do but vomit." Nausea and vomiting may both be absent particularly in retro-caecal appendicitis.

Constipation or Diarrhoea.

Constipation is the rule but diarrhoea may be present in children or in pelvic appendicitis with peritonitis.

(II.) Additional Features

These are present in virtue of the position the appendix adopts, e.g., in pelvic appendicitis, bladder symptoms, such as retention or incontinence of/

of urine, painful micturition or pyuria; or rectal symptoms such as pain in defæcation or diarrhoea may arise; while in retro-cæcal appendicitis symptoms like renal colic may be present.

CORRELATION OF SYMPTOMS WITH PATHOLOGICAL FINDINGS.

We have studied the various pathological conditions which may beset the appendix and its neighbouring structures and we have just considered the ordinary symptomatology of the disease. The next questions we have to ask ourselves are : "Can we correlate the pathological entities with the clinical picture? Do they run parallel ? Is the severity of the former reflected in the severity of the latter ?

The answer to the above can be gleaned from a study of the accompanying atlas where a description is given of the pathological findings together with the clinical history of the patient.

Case No. 9. whose pulse and temperature were normal actually walked into hospital although he had a large appendix abscess.

Again, Case No. 24. although temperature and pulse were normal, had a perforated appendix and general peritonitis.

In the other cases, the pathological findings and clinical history run more or less parallel. The moral is that the clinical features are not always to /

to be relied upon as giving a true picture of the pathological process especially in the obstructive type described by Wilkie.

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CHAPTER VI.D I A G N O S I S.

Robert Hutchison in introducing a communication on principles of diagnosis says, "It is written that there abideth Faith, Hope, Charity, these three, but the greatest of these is Charity. And so in medicine we have Diagnosis, which is a matter of Faith; Prognosis, which is a question of Hope, and Treatment which is only too often an affair of Charity: but the greatest of these is Diagnosis. For without accurate diagnosis it is impossible to forecast the course and outcome of a disease or to treat it satisfactorily."

There can be no doubt that this is true, for failure to recognise acute appendicitis when it is present may be followed by disastrous results to the patient. Diagnosis included

- (I) Actual diagnosis.
- (II) Differential diagnosis.

(I) ACTUAL DIAGNOSIS

We are told that 'diagnosis rests on three legs', viz., a knowledge of

- (a) History.
- (b) Symptoms.
- (c) Signs of any disease.

It is true that the diagnosis of acute appendicitis may be the simplest thing imaginable but, nevertheless, there are cases where it may constitute one of the most difficult/

difficult and entrancing problems of the clinician. Our conception of the disease was first placed on a scientific basis by the classical work of Reginald Fitz of Boston in 1886 when he showed that the symptoms were referable, not to perityphlitis, as it was then called, but to a pathological lesion of the vermiform appendix.

(a) History:

The patient may have had previous attacks of appendicitis (cf. cases Nos. 1. & 2. in accompanying atlas) or may have enjoyed perfect health until the onset of his present illness. Many attacks follow dietetic indiscretion and patients frequently give a history of constipation.

(b) Symptoms:

These have already been considered under the heading of Symptomatology, and diagnosis is to be arrived at by constructing a composite picture of the disease by grouping those individual symptoms and by correlating the times of their development. The three outstanding symptoms are pain, tenderness and rigidity, and we owe to J. B. Murphy of Chicago a description of the usual march of events. According to him, the symptoms, in order of time are (1) epigastric or umbilical pain (2) nausea or vomiting, (3) pain localised to right iliac fossa, (4) fever.

The first three are often referred to as Murphy's triad, and if tenderness and muscular rigidity in the right/

right iliac fossa are also present, then it is pathognomic of acute appendicitis.

Murphy has rightly emphasised the fact that if vomiting precedes the initial pain the disease is not one of appendicitis, but it is somewhat unfortunate that he should have included fever because it is my experience that many cases of perforated appendix have sub-normal temperatures. The absence of fever should never negative a diagnosis of acute appendicitis and failure of the medical attendant to recognise this fact has undoubtedly resulted in the loss of many lives.

While eliciting the symptoms, note should be made of any menstrual disorder, painful micturition, cough, etc., and most important of all - if, and when, an aperient has been taken; the relationship of aperient to perforation of the appendix will be emphasised later.

(c) Signs:

These are elicited from a clinical examination of the patient, which must be a thorough one, and includes a study of attitude, facial expression, together with examination of abdomen, chest, reflexes, throat, and urine.

First we note the attitude of the patient. In the presence of any acute abdominal disease, he prefers to sit up with his knees flexed; and much may be learned from his facies; It is difficult to describe it accurately, but one versed in clinical experience can readily tell from the facial expression whether serious/

serious pathological processes are at work. The drawn expression, the clear anxious eye, the circum-oral pallor, all betray signs of acute illness. I also lay great stress on the condition of the tongue. In the worst forms of appendicitis it is dry, covered with a brown fur, and the breath emits a faecal odour. Professor Gulland used to classify patients suffering from pneumonia into (1) those in whom the tongue was moist where the prognosis was good and (2) those whose tongue was dry where the prognosis was more grave. I believe even greater truth could be attached to such a classification of patients suffering from acute appendicitis. A dry tongue means paralysis of the salivary glands due to absorption of peritoneal toxins.

The importance of the faecal odour of the breath was impressed upon me some years ago while administering anaesthetics. Two cases of strangulated herniae and one case of acute appendicitis were being operated upon on the same evening. All three had faecal odour of the breath and all three had peritonitis. Since then, I have frequently been able to confirm the observation that a faecal odour of the breath, in cases of appendicitis, means involvement of the peritoneal cavity.

Again, I have observed that the first sign of aberration from the normal in acute disease is the loss of a sense of humour, and its return is almost the earliest evidence of convalescence. In my lectures/

lectures to the nurses I point out that a dry tongue and absence of a smile after operation denote grave prognosis; while the presence of a moist tongue and a smile signify recovery from acute disease.

Abdominal examination.— I usually begin by asking the patient to point with one finger to the site of maximum pain - this being specially valuable in children. Inspection may reveal diminished movement of the muscles in the right iliac fossa on respiration, with tenderness and rigidity localised to the right iliac fossa and possibly hyperaesthesia. These, if present, usually denote localised acute appendicitis. Extension of tenderness and rigidity with dulness on percussion, or distension of the abdomen signify the presence of spreading peritonitis.

The presence of local swelling and dulness in the right iliac fossa may mean an appendix abscess, an appendix surrounded by omentum or both.

Dickinson has tried to attach diagnostic significance to the various points of maximum intensity, and although I cannot agree with his deductions, I append his statements :-

"In chronic appendicitis, Morris's point is always tender, Monroe's somewhat so, McBurney's never.

In sub-acute appendicitis with hyperplasia, Morris's point is tender, Monroe's and McBurney's equally so.

In acute appendicitis, the first condition is reversed. McBurney's point is tender, Monroe's at times, and Morris's never, unless, (as not uncommonly happens) there be an acute attack on top/

top of a chronic one, when there will be tenderness at both Morris's and McBurney's points."

It is necessary before completing the examination of the abdomen to carry out certain tests to exclude various positions of the appendix. The importance of a rectal or vaginal examination to exclude pelvic appendicitis has already been emphasized and, in all abdominal disorders, we would do well to remember the dictum of the late Professor John Chiene, viz. "examine the rectum."

The obturator test (the eliciting of pain in the right lower abdomen on full internal rotation of the flexed thigh) may be present in cases of pelvic abscess, and results from stretching an oedematous obturator internus muscle.

The psoas test (pain in the right iliac fossa upon flexion or extension of the corresponding thigh) usually denotes a retro-cæcal appendicitis; in one case where the test was positive, the appendix was found at operation to be buried in the substance of the psoas.

Examination of the chest should never be omitted. This is important as the pains associated with rheumatic fever or the referred pain of pneumonia may closely simulate the "acute abdomen." This applies particularly to children.

While examining the bases of the lungs, advantage should be taken of the opportunity to percuss the spinal column to exclude the possibility of Pott's disease with referred pain being mistaken for appendicitis./

appendicitis.

Also renal tenderness should be looked for and examination of the urine will exclude lesions of the genito-urinary tract and diabetes mellitus.

Finally the throat should be inspected; and tabes dorsalis excluded by testing the knee and ankle jerks. Only by this complete examination can conditions which simulate appendicitis be differentiated.

(II) DIFFERENTIAL DIAGNOSIS

In discussing the differential diagnosis and complications of typhoid fever, Osler used to cover two thirds of his lecture course on medicine, but I believe I would have to refer to two thirds of both medicine and surgery if I were to mention all the diseases from which appendicitis might be differentiated.

Within the last three years I have found the following diseases erroneously diagnosed acute appendicitis - the number of such cases being given in brackets -

(1) Extra-Peritoneal Conditions.

- | | | | |
|-----|--------------------|-------------------|-----|
| (a) | Supra-abdominal | espec. pneumonia | (8) |
| | | and diaphragmatic | |
| | | pleurisy .. | (2) |
| (b) | Infra-abdominal | e.g. Gonococcal | |
| | | Epididymitis | (1) |
| (c) | Anterior abdominal | Neuritis | (3) |
| | wall | Diabetic neuritis | (1) |
| | | Rheumatic fever | (3) |
| (d) | / | | |

- | | | |
|-----|---------------------|--------------------|
| (d) | Posterior abdominal | Pott's disease (2) |
| | well | Tabes dorsalis (1) |

(2) Intra-Peritoneal Conditions.

- (a) Colics:- intestinal (6); renal (12);
biliary (4); lead (1); tubal (1).
- (b) Inflamm- pancreatitis (1); cholecystitis (4)
ations:-pyelitis (5); appendagitis (14).
- (c) Perfor- peptic ulcer (7); gall-bladder (2)
ations:- ectopic gestation (2).
- (d) Intestinal
obstruction: (6)[One was case of Meckel
Diverticulum] .
- (e) Torsion of Ureter with Diett's crisis (5)
Pedicles :- Twisted ovarian cyst (1).

To confuse acute appendicitis with any other condition included in the 'acute abdomen' is usually not a serious mistake, as the correct diagnosis is practically always revealed at operation. But there are other conditions which may simulate acute appendicitis for which the ordinary abdominal operation is not only useless but definitely contra-indicated. I wish to give clinical histories of what I have found to be the three most common of these.

(i) Pneumonia.

It goes without saying that a patient suffering from pneumonia should not be subjected to exertion let alone administration of anaesthesia for the removal of a normal appendix. The mistake usually results from failure to consider pneumonia as a possible differential cause, and from neglect to examine the chest as a routine. Chief difficulty occurs in children; /

children; in several cases I have found pneumonia and desisted from removing the supposed pathological appendix:ix. I have notes of one case, however, in which operation was performed. The patient was admitted with a diagnosis of perforated duodenal ulcer. On examinations his rigidity was chiefly in the right iliac fossa; one of my colleagues operated and found only a slight serous fluid in the peritoneal cavity. Next morning definite signs of consolidation at the right base was detected and a diagnosis of pneumonia made. Unfortunately the patient died. This illustrates what fatal results may follow a confusion of these two conditions.

(ii) Rheumatic fever.

I have notes of three cases of rheumatic fever which were admitted to hospital as acute appendicitis.

- (a) J.B.J. aged 12 years, complained of abdominal pain and vomiting; T. 101.6°: P. 124: R. 28. Abdominal tenderness and rigidity were present all over the abdomen. Doubt was expressed as to the diagnosis but it was considered wiser to operate. Operation revealed an excess of serous fluid in the peritoneal cavity but the appendix was normal. The following day pain and swelling of both ankle joints suggested the correct diagnosis of rheumatic fever.

It is interesting to note that this is the only case I know of where predilection of the micro-organism of rheumatic fever for endo-thelial structures seemed to involve the peritoneum.

- (b) A.J. aged 17 years - admitted as sub-acute appendicitis. His only symptom was pain over the right iliac fossa; no vomiting; no tenderness/

tenderness; no rigidity; T. 100 : P. 90 : R. 20. Operation not performed. Three days after admission a faint mitral-systolic murmur suggested the diagnosis which was corroborated by subsequent development of rheumatic nodules and auricular fibrillation.

- (c) B.E.T. aged 23. - admitted as acute appendicitis. T. 103.8° : P. 128 : R. 22. Complained of pain in right loin; vomiting; slight tenderness; but no rigidity in right iliac fossa. High temperature suggested pyelitis but the urine contained no abnormalities. Marked follicular tonsillitis and mitral systolic murmur made the diagnosis of rheumatic fever an obvious one. Operation was not performed, the temperature and symptoms subsiding after large doses of sodium salicylate.

These cases illustrate the importance of examination of the throat and heart; and show that involvement of the peritoneum and the sheaths of the abdominal muscles in rheumatic fever may simulate acute appendicitis.

(iii) Renal cases.

These constitute my chief difficulty in the differential diagnosis of acute appendicitis. I have had the strange experience of examining ten successive cases diagnosed as acute appendicitis and of finding in all various renal conditions; the last patient in the series had sub-acute appendicitis and calculous pyonephrosis of the right kidney.

In the last six months I have had to correct the opposite mistake; several cases being admitted as renal colic and found to have retro-caecal appendicitis. The difficulty of distinguishing between reno-ureteric lesions and retro-caecal appendicitis is a very real one./

one. In my present position as urologist in this hospital, I am constantly meeting this difficulty; the presence of an abscess in the right loin with a normal urine may result either from a retro-caecal appendix abscess or from a perinephric abscess originating in a "closed pyonephrosis" where the urine is excreted only from the healthy kidney. In such cases I perform a preliminary cystoscopy; if both ureteric orifices are normal and efflux of indigo-carmin appears simultaneously from both orifices, then I proceed to operate as for retro-cæcal appendicitis. The performance of cystoscopy has often been the means of revealing renal lesions which were diagnosed as appendicitis and vice versa.

Mention must now be made of the difficulties of diagnosis in (a) children (b) women of child-bearing period.

(a) Children.— John Fraser (Edinburgh) in discussing the abdominal emergencies of childhood points out that the clinical examination and treatment of disease in children, compared with that in adults, presents certain features which are favourable to the clinician and others which are unfavourable.

Examination and diagnosis are easier because

- A. lesions are restricted to the ileo-caecal angle or centre of the abdomen; upper abdominal or pelvic conditions are rare.
- B. limited pathology - malignant disease, chronic ulcerative conditions or perforations are rare - the type of lesions being limited to (1) acute inflammations (2) tuberculosis (3) mechanical disorders./

disorders.

- C. Psychological factor.— The clinician can trust absolutely the child's subjective phenomena; he has not yet acquired the art of dissimulation.

Unfavourable features include

- A. difficulties of examination. If the child cries or strains very light anaesthesia should be administered when tenderness and muscular rigidity can still be elicited.
- B. Children have a very narrow 'safety threshold'; they go downhill rapidly: diagnosis must be made early and treatment instituted immediately.

The above remarks can well be applied to children in whom acute appendicitis is suspected.

Among the conditions which may be confused with appendicitis in childhood are the following :—

- (1) Gastro-enteritis.
- (2) Pneumonia.
- (3) Pyelitis (especially in females).
- (4) Intersusception (especially in males).
- (5) Cyclical vomiting.
- (6) Tuberculous ileo-caecal glands.
- (7) Pneumococcal peritonitis (especially in females.)

Routine examination will exclude Nos. (2) and (3) while Nos. (1), (4), and (5) are not usually difficult or common.

I wish to refer in particular to conditions No.(6) and No. (7).

Tuberculous glands.

These are common in the ileo-caecal region and in the mesentery, and may give rise to symptoms practically identical with those of appendicitis. Struthers of Edinburgh writes : "As a rough illustration of the relative/

relative frequency of the condition I may state that in the two years 1919 and 1920 I met with 22 cases of mesenteric lymphadenitis while during the same period I dealt with 187 cases of appendicitis. In children and adolescents lymphadenitis is more often confused with appendicitis than with any other disease."

Carson of London has drawn attention to the sudden onset and sudden cessation of the pain; and McFadden states that the pain (in lymphadenitis) may begin at the umbilicus and settle in the right fossa - the attacks of pain usually being preceded by headache and drowsiness. Vomiting is a frequent symptom both before and after the pain. He shews that the site of tenderness in inflammation of the ileo-caecal glands is not McBurney's point but a situation higher up and more medial than this point.

Miss Herzfeld (Edinburgh) states that abdominal tenderness may be very marked but rigidity less so, and the child on the whole was not so ill. This condition might be very difficult to distinguish from appendicitis and in doubtful cases it was better to explore.

My own experience has shown that the two conditions may be quite indistinguishable clinically and operation has revealed sub-acute (or chronic) appendicitis in association with enlarged ileo-cæcal glands. I have never been able to satisfy myself as to the real cause of the glandular enlargement - whether secondary to/

to appendicitis or primarily tuberculous. It is my custom to remove the appendix and during convalescence to prescribe general treatment as for tuberculosis together with tuberculin injections. So far as I can judge the results are satisfactory.

The differential diagnosis, then, between lymphadenitis and appendicitis is notoriously difficult but, as Struthers puts it, "Appendicitis is a treacherous disease, and in doubtful cases a 'wait and see' policy is apt to be followed by disastrous results especially in children."

Hence operation in the majority of cases is the wiser course.

Pneumococcal peritonitis.

I have met with four such conditions in the past three years. All were female patients between five and nine years of age and all four died. In three of them a correct pre-operative diagnosis was made. The first child, F.M.J. aged 6 years, was admitted with "acute appendicitis and chronic bronchitis"; frequency of micturition and diarrhoea had been present for two days; also abdominal pain and vomiting; T. 103.8° : P. 138 : R. 36. Examination shewed marked cyanosis with pain, tenderness and rigidity of whole abdomen especially below the umbilicus where there was distension. Scattered areas of consolidation were detected in both lungs and rectal examination revealed tenderness of pelvic floor.

Typhoid/

Typhoid, general peritonitis complicated by pneumonia, pneumococcal peritonitis were the possible conditions considered. The last, because of age, sex, urinary symptoms, was most likely. Operation was contra indicated as the child was in extremis; Murphy-Ochsner treatment was instituted but child died 36 hours after admission. Post-mortem examination revealed abdomen full of pus and flaky lymph over the intestine. Bacteriological examination shewed numerous pneumococci but the vaginal swab was negative.

The other three cases were not quite so ill on admission. Operation revealed pelvic peritonitis; a drainage tube was inserted into the pelvis but all subsequently developed pneumonia, pneumococcal septicaemia and died.

Fraser and McCartney, Edinburgh, have investigated this condition and they emphasise the following points:

- (a) The disease occurs in female children
 - (1) Age period is from 2-10 years with maximum occurrence at 5th year.
 - (2) Incidence practically confined to the poorer classes.
- (b) The micro-organism gains access through the Fallopian tubes to the pelvic peritoneum. The age incidence is explained by the work of Professor Arthur Robinson of Edinburgh who has shewn that the vagina becomes patent about the 5th year of life.
- (c) They recommend early operation for drainage (under gas and oxygen never ether or chloroform) and blood transfusion in cases of septicaemia.

In all acute abdominal diseases in female children between/

between five and seven years of age, I regard pneumococcal peritonitis (primary) to be the most likely diagnosis.

(b) Women

The following conditions are chiefly to be considered :-

- (1) Pyelitis - especially during pregnancy.
- (2) "Appendagitis", viz. salpingo-oophoritis.
- (3) "Subinvolution peritonitis".
- (4) Ectopic gestation.

(1) Pyelitis

Eight cases of "pregnancy and appendicitis" were admitted and in seven of them the symptoms were due to pyelitis of pregnancy. (Pyelitis, like typhoid fever, always seems to be overlooked.) The diagnosis, in these cases, was quite easy; the patients were in the sixth - seventh months of pregnancy; there was no abdominal rigidity; the urine was acid, contained pus and albumen; and pyrexia was present. The alliteration which I give in my lectures to the nurses regarding this condition sums up the differential points - 'pyrexia with pyuria in pregnancy is practically pathognomonic of pyelitis and the proper physic is potassium citrate.'

Pregnancy may co-exist with appendicitis as was shewn by the history of the eighth patient. However, she was only three months pregnant. (pyelitis is commonest at the seventh month). A catheter specimen of urine shewed no abnormalities; and abdominal tenderness/

tenderness and rigidity were present in the right iliac fossa. A diagnosis of acute appendicitis was made and at operation I removed a gangrenous appendix which had perforated. Fortunately the patient recovered and abortion, though anticipated, did not take place.

(2) "Appendagitis"

This term I use to include inflammation of the tubes and ovaries.

This may be very difficult to distinguish from appendicitis. Formerly, I used to regard tenderness in both iliac fossae to be in favour of a diagnosis of bilateral appendagitis, but the fallacy of this assumption was proved by the following case.

Mrs. F. complained of abdominal pain and vomiting; T. 100.2° : P. 100 : R. 20. On examination I found tenderness in both iliac fossae and on pelvic examination in the pouch of Douglas. I regarded the case as one of ascending infection but at operation a perforated pelvic appendix was removed.

This illustrated to me, in an impressive way, that pelvic appendicitis may be accompanied by bilateral abdominal tenderness.

Kelly, in his classical monograph, lays stress on this point and states that: "Hypogastric pain with tenderness and rigidity on both sides is very characteristic of pelvic appendicitis."

As already stated, appendicitis and appendagitis frequently co-exist and, in all cases of doubt, operation/

operation is the wisest course to follow.

(3) Subinvolution peritonitis.

This is the term suggested by a writer in the British Medical Journal for a symptom complex which will be more easily explained by giving a short clinical account of such a case.

Mrs. L.M.E., aged 23 years; first confinement 4 weeks ago; forceps delivery. Two days ago, complained of abdominal pain; repeated vomiting; aperient taken 30 hours ago; T. 104.8° : P. 118 : R. 28. On examination slight infra-umbilical rigidity. Temperature seemed higher than usual for acute appendicitis but because aperient had been taken, operation was performed. This revealed

- (i) glairy fluid in the peritoneal cavity.
- (ii) a boggy, enlarged, subinvoluted uterus.
- (iii) congestion of the lower end of the ileum and appendix.

The patient recovered after drainage.

A fortnight after the above operation I read a letter in the British Medical Journal (about Sept. 1927) giving an account, identical to the one quoted above. The writer suggested the term "subinvolution peritonitis" and he regarded the condition as being due to infection spreading from the uterus to the peritoneal cavity.

Since then I have had two more cases; one, a fortnight after confinement, the other, three weeks after a miscarriage.

(4) Ectopic gestation.

I have notes of two cases of ruptured ectopic gestation which were operated upon for 'acute appendicitis.' In both, no history of menstrual disorder was obtained. Pain and rigidity in right iliac fossa were present with dulness in the supra pubic region. Pelvic examination shewed swelling in the right side of the pelvis which was considered an appendix abscess. Both patients recovered and it is interesting to state that the Zondek-Asscheim test was positive.

From the above discussion it will be seen that acute appendicitis may mimic numerous diseases and lends truth to the well-known dictum that in any acute abdominal disorder we should "think first, last and always of appendicitis" (Deaver).

Again, as the appropriate line of treatment for these diseases differs widely from that employed for appendicitis, we are forced to agree with Hutchison when he says "The first part of treatment is diagnosis, the second, diagnosis, and the third, diagnosis."

There are three questions which the surgeon wishes to answer from his examination of the patient.

(1) Is it appendicitis ?

A knowledge of the symptoms, and their time relationship as set forth in this and the preceding chapter, together with the results of a routine examination, should form a satisfactory answer to this question.

(2) If so, what is the position of the appendix ?

Attention to the areas of localised rigidity and tenderness; and the use of rectal or vaginal examination; obturator test; and psoas test should provide the required/

required answer.

- (3) Has the peritoneal cavity become involved ?

Anxious facial expression, dry tongue, with faecal odour of the breath; and spreading tenderness and rigidity will make the presumption of spreading peritonitis a safe one.

And lastly, if satisfied by diagnosis that the appendix is involved, the surgeon must act upon that diagnosis, and proceed immediately to carry out some form of treatment. Zachary Cope states that in any

acute abdominal illness there should be a time limit to diagnosis. Further, there should be no procrastination of treatment for "procrastination is the thief of lives."

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CHAPTER VII.THEPROBLEMOFCHRONIC APPENDICITIS

The problem of chronic appendicitis has long confronted the medical profession; like the poor, it is always with us, and there seems to be little likelihood of immediate relief from its perplexities. It constitutes, in fact, not one problem but a veritable host of them. It presents debatable criteria from all points of view; in its aetiology and pathology, as well as in its diagnosis and treatment, it leaves much to be desired.

In my opinion it is in the treatment of chronic disease -- both medical and surgical -- that further research and progress are required. The early application of the surgical art to acute abdominal affections such as strangulated herniæ, perforated gastric and duodenal ulcer, etc., has saved thousands of lives and has brought a return of health and happiness to patients, in a manner both speedy and spectacular. It is only natural, therefore, that the surgeon should be keenly interested in the problems of the acute abdomen; Is he equally interested in those vague chronic disorders of the human body which make patients "not well enough to work, and yet not ill enough to remain in bed" ?

The/

The greatest number of surgical failures centre around the chronic abdomen and the time is ripe for a more scientific consideration of the problems which it presents. This applies particularly to the problems of chronic appendicitis.

I venture to suggest that there are few diseases concerning which so many different opinions are held, as that of chronic appendicitis.

First, as regards aetiology: Is the chronic appendix always a legacy from some previous attack, or can the condition originate de novo ?

Some, like Trotter, are agreed that it may originate as a primary chronic appendicitis; while, in America, a large majority regard the condition as being a resultant of an acute attack. Some even go so far as to say that there is no such thing as chronic appendicitis. Walton, while taking part in the discussion on this subject at the British Medical Association Meeting held at Edinburgh, presents statistics which, he states "strongly support the view that chronic appendicitis does not exist as a primary disease, but only occurs after an acute attack," and he refers to the dictum formulated by Willys Andrews that a chronic appendicitis should never be diagnosed unless there has been an acute attack.

The concensus of opinion seems to lie between those two extremes; it is agreed that while in the majority of cases the condition is an aftermath of an acute/

acute attack, yet there are instances in which it would seem to originate as a chronic form.

Again, the obliterative processes seen in late adult life are ascribed by some to involutionary processes (Ribbert), while others regard them as being due to the results of inflammation (Berry).

Then, as regards pathology, it seems to be difficult to define the appearances of the chronic appendix. What some would regard as pathological, others would regard as normal. Fraser speaks of surgeons removing appendices, and donning a convenient pair of pathological spectacles to describe lesions and abnormalities in what are really healthy looking organs.

Many observers state that the lesions characteristic of the condition are visible only on microscopic examination while others hold that a diagnosis can be made macroscopically.

I have already described from sections depicted in the accompanying atlas what I regard to be the most common pathological findings in these cases, viz., a thickening of the walls especially involving the submucous coat; with stricture, kinking or angulation and obliteration of the lumen; and adhesions binding down the peritoneal coat.

Again, considerable difficulty is experienced with regard to the actual and differential diagnosis. This arises from the fact that the chronic appendix is protean in its manifestations. Its symptoms may simulate/

simulate deranged function of practically any abdominal viscus and may also lead to confusion with lesions of the kidney and ureter; the spine; the sacro-iliac and hip joints. The most common error, however, proceeds from failure to appreciate that pain originating in the right iliac fossa is not usually due to appendicitis. Sherren and Morley have already been quoted as pointing out this fallacy; while Fraser in a recent article on "Pain in the iliac fossa" describes "an extensive summation of nerve tissue in this comparatively small area which may be regarded clinically as indicating a wide potential source from any point of which there may be an ultimate superficial iliac pain." He mentions among others the following conditions which are to be differentiated from chronic appendicitis:

Spasm of the ileo caecal junction,
Oedema of the psoas muscle, and
Irritation of the lumbar and sacral nerves
by osteophytic outgrowths around the
intervertebral foramina.

Carson of London points out that, apart from a definite history of a previous attack, the only reliable way of diagnosing chronic appendicitis is from the presence of appendicular dyspepsia.

I have seen many patients giving a history of this condition. They complain of epigastric pain and vomiting, usually after meals, and tenderness can be elicited in the epigastrium and, to a lesser extent, in the right iliac fossa; but pain is never complained of/

of over McBurney's point.

There is no definite rhythm of symptoms as is found in gastric and duodenal lesions. Moynihan and Paterson were among the first to draw attention to this condition of appendicular dyspepsia and the former has described three stigmata at operation which enable him to establish the diagnosis of chronic appendicitis without ever seeing the appendix. These are

- (1) congestion of the pyloric portion of the stomach (the pyloric blush),
- (2) spasm of the pylorus, and
- (3) enlarged glands in the great omentum.

The dyspeptic symptoms produced by an appendix make differentiation from gastric ulcer, duodenal ulcer, and disease of the gall-bladder difficult. It was Moynihan who said that most gastric ulcers are found in the right iliac fossa ! But co-existing lesions of those organs may be found and constitute what is called the abdominal triad. Wilkie, Moynihan, Braithwaite and others have shewn that peptic ulcer and cholecystitis may result from infection originating in the appendix. Braithwaite suggests that this is carried via aberrant and retrograde lymphatic flow; Wilkie and Moynihan favour spread by the blood stream.

In my opinion, the condition which is most frequently mistaken for appendicitis is renal or ureteric colic. In the section on differential diagnosis of acute appendicitis, I have already emphasised the importance of performing cystoscopic examination to differentiate/

differentiate between retro-caecal appendicitis and renal colic; and this necessity is all the more imperative in cases of so-called chronic appendicitis. I usually carry out two preliminary tests in any adult admitted to hospital with the diagnosis of chronic appendicitis before operating (1) a Barium meal to exclude associated upper abdominal lesion; (2) cystoscopy with, if necessary, ureteric catheterisation and pyelography.

I am convinced that it is only by so doing that an accurate diagnosis can be arrived at, with a corresponding diminution in the number of unsuccessful appendicectomies. I have had several cases of mobile kidney with intermittent hydronephrosis in women who had previously had their appendix removed without relief of their symptoms; and again, I have found five cases of ureteric calculus which had been wrongly diagnosed as appendicitis. I have notes of one instance, however, illustrating the opposite mistake:

A.J.F. aged 29 years, complained of pain in the right hypochondrium with bouts of vomiting. X-Ray examination shewed a shadow in the line of the ureter. Ureteric catheterisation was not performed. At operation no calculus was found in the ureter but a faecolith corresponding in size and shape to that seen in the radiogram was found in the appendix.

These cases illustrate the necessity for cystoscopic and allied examinations.

I should like to draw attention to a condition which I have not yet seen described, viz., constriction/

constriction of the ureter due to adhesions following acute appendicitis. I have notes of two such cases, while Mr. T. E. Hammond, Urologist at Cardiff Royal Infirmary, (in a personal communication) gave me details of a third. This occurred in a doctor whose appendix had been removed three years previously for acute retro-caecal appendicitis. He was free from symptoms for two years after the operation when he complained of pain in the right loin - no abnormalities being found in the urine. The patient pleaded for a pyelography. Mr. Hammond agreed but said he did not think any improvement would result. During examination the ureteric catheter became obstructed at 10 cms. but a smaller size could be passed up to the right kidney and later larger catheters. From that day the patient's symptoms entirely disappeared.

I have had two cases identical to the above; in all, the appendix had been acutely inflamed and retro-caecal in position. Presumably, adhesions had contracted the ureteric lumen and the symptoms were relieved by dilatation with ureteric catheters.

In women, the task of distinguishing between appendicitis and appendagitis is a very difficult one. In most cases I am content to say that either or both may exist, the actual lesions being dealt with at operation. I have been astonished at the frequency with which symptoms of premenstrual dysmenorrhœa have been proved by operation to be due to a chronic appendix/

appendix adherent to the ovary and tube.

Stratham, in discussing the influence of the vermiform appendix on gynaecological surgery, comes to the following conclusions :-

- (1)"The appendix is very likely to be affected by pelvic inflammations and may be the cause of salpingo-oöphoritis and its consequences more frequently than is expected.
- (2) The appendix should be suspected and examined in all cases of pelvic peritonitis, and it should be removed if it is in any way diseased, to prevent further trouble at a later date.
- (3) Even when the operation is performed for a condition which is not inflammatory the appendix should be examined.
- (4) Acute pyosalpinx is frequently complicated by acute appendicitis and vice versa.
- (5) I would advance with great diffidence that it is just as well to remove a pelvic appendix whenever found, even if it appears healthy to the eye."

In children the chief differential diagnosis is tuberculosis of the ileo-caecal glands. Chronic appendicitis does occur in children (although much less frequently than acute appendicitis) and differentiation is often possible only at operation.

In elderly people the possibility of emaciation being due to chronic appendicitis should be kept in mind. I have had two cases where malignant disease was suspected and where operation revealed chronic obliterative appendicitis, the patients putting on weight after appendicectomy.

Recently Mitchell of Belfast has been going so far/

far as to regard cases of migraine and epilepsy as due to "appendix reflex"; and he quotes cases improving or being cured after appendicectomy.

The appendix has also been regarded as a focus of sepsis and I have seen one patient who had merely drainage for an appendix abscess develop septic arthritis of the spine, the arthritic symptoms being arrested after appendicectomy.

Another patient who had had an appendicectomy nine months previously complained of excruciating pain over the lumbar spines - X-Ray examination shewed nil abnormal. I suggested the diagnosis of typhoid spine which was confirmed by subsequent improvement and by a positive Widal reaction (agglutination reaction to *B. paratyphosus* beta in dilution 1:250). I have no doubt that the correct diagnosis of the previous condition for which appendicectomy was performed was typhoid fever.

X-Ray Diagnosis:

Even as regards the value of X-Rays in the diagnosis of chronic appendicitis, great diversity of opinion exists. Bertwistle, for example, states that "radiology taken in conjunction with clinical signs and symptoms probably gives us more definite assistance in diagnosis than any other means." If the appendix is visible he regards the following appearances as suggestive of disease :

- (1) An irregular lumen. While some parts are dilated, others are constricted. Especially/

Especially important, according to Spriggs and Marxer, is a claw-shaped termination of the barium column.

- (2) Tenderness over the appendix. Here one can actually place a finger over the organ.
- (3) Concretions. These appear as mottled areas interrupting the barium column. If large and stationary over several days, they must be looked on as evidence of defective musculature. (Occasionally they become impregnated with lime and cast a shadow mimicking a ureteral calculus.)
- (4) Spasm. This shows itself as one or more abrupt terminations in the opaque column. It is usually associated with ulceration.
- (5) Acute kinks. These may be congenital or the result of adhesions.
- (6) Spasmodic conditions of stomach, duodenum, and colon. These are commonly found in chronic appendicitis, but are also found in other abdominal conditions.

Orliansky states that the appendix, after purgation, can be visualised radiologically in 90 per cent. of cases; he regards a dilated appendicular canal, and particularly the club-like dilatation of the distal end of the appendix as almost certain signs of a diseased appendix. He does not attach much importance to tenderness of the visualised appendix in the absence of other confirmatory signs of a diseased appendix, and he states that the non-filling of the appendix can be interpreted as a pathological sign only in cases when the appendix cannot be visualised after repeated purgation, each purgation being followed by a Barium meal.

Hurst/

Hurst urges that X-Rays afford the greatest help to the diagnostician, the appendix being visible in 100 per cent. of normal people and in 80 per cent. of people with chronic appendicitis; he emphasises the importance of eliciting localised tenderness under screen examination, and he says that "in all suspected cases inflation of the colon should be performed, combined with X-Ray examination, and, unless one or both of these were definitely positive, a diagnosis of appendicitis should not be made."

Others, more sceptically minded, regard X-Ray examination as of little or no value. Carson considers tenderness over a visible appendix as of no import whatever, because between the appendix and the leaded glove is the eleventh nerve entering the sheath of the rectus.

Wilkie states that X-Rays are of value for shewing an associated lesion in the stomach or duodenum, but for the diagnosis of chronic appendicitis they are useless.

It will be seen that the diagnosis of chronic appendicitis is never a very definite one. It is largely a diagnosis by elimination and in contrast to acute appendicitis in which there should be a definite time limit for diagnosis, a most exhaustive examination extending perhaps over a period of several days is necessary before a diagnosis of chronic appendicitis can be made.

TREATMENT/

TREATMENT:

Since the diagnosis of chronic appendicitis leaves much to be desired this is reflected in the unsatisfactory results which follow appendicectomy. John Fraser, while discussing the disturbances of the involuntary nervous system, shews how closely ileo caecal pain might resemble appendicitis. He says: "During six months' work in the Edinburgh Royal Infirmary, I have kept a careful record of fifty-eight cases sent to hospital with the diagnosis of appendicitis. Twenty-four of these showed pathological conditions of the appendix; thirty-four were examples of ileo-caecal pain certainly, but the explanation was not an inflammatory lesion of the appendix, but a spasm of the caecum and the related colon. There was a diagnostic error which exceeded 50 per cent."

Many American surgeons have emphasised the number of needless appendicectomies, and my own experience has convinced me that even when operating for chronic appendicitis, an exploratory laparotomy allowing examination of other abdominal viscera should be undertaken. This was brought home to me by the following case :-

T.A. aged 25 years was admitted as a case of perforated duodenal ulcer. Eight months previously I had removed his appendix by the McBurney incision. I have no doubt that this ulcer would have been discovered if an incision to allow of investigation of the stomach and duodenum had been used.

One of the chief points in operating upon the chronic/

chronic appendix is the use of a good incision and I strongly advocate the following procedure.

1. In a child, as the chief differential diagnosis is tuberculous ileo caecal glands, a McBurney incision is all that is necessary.
2. In the adult male, exploratory laparotomy by a right supra umbilical paramedian incision is indicated to exclude associated peptic ulcer.
3. In the adult female, either (a) an infra umbilical paramedian incision should be used to exclude lesions of the pelvic viscera or (b) a right supra umbilical paramedian incision to exclude an associated gall-bladder.

Burgess of Manchester lays great stress on this aspect; he says: "The comparative ease, safety and success with which, nowadays, quiescent appendicectomy can be undertaken has led to its performance on a very large scale - often on very insufficient evidence that the appendix is the real cause of the symptoms complained of. I feel very strongly that the steadily increasing number of patients coming into our hospitals and nursing homes with chronic abdominal disease, upon whom a more or less recent appendicectomy has been performed without material relief to the symptoms, constitutes a very serious reproach to abdominal surgery. The appendicectomy qua appendicectomy has been skilfully performed, the wound has healed primarily, without complication, and yet, the symptoms not having been relieved, it is obvious that the appendix was not the cause of the trouble, or at any rate, not the sole cause. The incision has usually been of the "gridiron type",/

type", but sometimes Battle's or other variety of limited incisions has been adopted. The fault lies in the fact that no general examination of the abdominal viscera was made during the operation, nor is such possible through any of these limited incisions; so far as non-urgent conditions are concerned, "grid-iron" appendicectomy is the curse of abdominal surgery, and there should be but one incision for this type - the "paramedian". This reproach to our work will never be removed until surgeons generally come to regard every abdominal section for non-urgent conditions as essentially and primarily an exploratory laparotomy, however certain they may feel of their pre-operative diagnosis. It is the opportunity of a life time to discover the state, not only of the organs suspected, but of all the other abdominal and pelvic viscera; nor should the abdomen be closed until the surgeon knows the condition of every viscus it contains, at any rate so far as inspection or palpation can reveal it."

To sum up: It is only by an extensive clinical examination aided by Barium meal, cystoscopy, pelvic examination and by an exploratory laparotomy to allow of examination of all the abdominal viscera that the problem of chronic appendicitis can be satisfactorily dealt with. Otherwise the operation of appendicectomy will be brought into disrepute and injure its use in acute appendicitis; and only thus will the pathetic clinical/

clinical picture drawn by Robert Hutchison be avoided. He describes patients first having their appendix removed, then the ovary, while at subsequent operations the kidney and gall-bladder are explored. The patient is utterly neurasthenic and until more care is taken in the diagnosis of chronic appendicitis, the outlook will, in the words of Hutchison, "be indeed a bleak one".

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for
CHAPTER VII

(CHRONIC APPENDICITIS)

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CHAPTER VIIIP R O G N O S I SandT R E A T M E N T.

These two aspects of the condition are so closely interrelated that it is appropriate to consider them together.

PROGNOSIS.

It was a cynical teacher of mine who, in his lectures, said: "There is one thing about your prognosis, gentlemen, and that is that it is usually wrong."

This rather pessimistic view scarcely seems warranted but it possesses the merit of drawing attention to the difficulties in giving a prognosis. Robert Hutchison in referring to this aspect says : "Of the three great divisions of practical medicine - diagnosis, prognosis, and treatment - prognosis is certainly the most difficult. It demands as much knowledge and power of observation as diagnosis, and even more experience and judgement. Experience, indeed, is, perhaps, what the prognostician requires most of all."

Prognosis consists of

- (1) Immediate prognosis - as regards immediate danger to life.
- (2) Ultimate or late prognosis, which concerns subsequent health.

In each of these, our opinion is influenced by certain/

certain general considerations, also by the collected results of our experience shown in statistical form, although Moynihan has issued a caveat against being too much influenced by the latter in his well known dictum, "Statistics can be proved to mean anything even the truth !"

(1) Immediate prognosis.

This will be influenced by the following factors :-

- (a) Early or late diagnosis.
- (b) Age and sex of the patient.
- (c) Type of appendicitis, its bacteriology and position.
- (d) Treatment adopted.

(a) Diagnosis.—

It is by improved and early diagnosis that patients can be operated upon before extension of the pathological processes from the appendix itself to the adjacent peritoneal cavity.

In as much as death results from the complications of appendicitis, chief and most dreaded of which is peritonitis, the mortality rate will only be lowered in so far as patients are operated upon before peritonitis supervenes. As the late Joseph Adams puts it : "If the mortality of appendicitis is to be abolished by treatment of the disease, sepsis must be eliminated, and the problem becomes one of early diagnosis. Diagnosis will always be the highest art both in surgery and in medicine, and let us recognise that herein lies the chance of practising preventive surgery which alone can bring down the mortality of this/

this disease to a level consistent with the pride of our profession."

Prevention of peritonitis must ever be the keynote to the treatment of appendicitis and in its presence a guarded prognosis should be given.

In my own series, 17 deaths occurred in 300 acute cases; one occurred where the inflammation was limited to the appendix and was due to the sister in charge giving an aperient on the second day after operation, thereby causing extroversion of the appendix stump, and peritonitis. Three deaths resulted from cases of appendix abscess while the remaining thirteen resulted from general peritonitis. The greater the involvement of the peritoneal cavity, the higher the mortality.

(b) Age and sex.—

Although the disease in its acute form is most common - according to my experience - between the ages of 10 and 30, it is at the extremes of life that the highest death rate is found. This is shewn by a study of the following table which gives an analysis of my own series of 300 cases.

TABLE/

TABLE

<u>AGE</u>	<u>NO. of CASES</u>	<u>PERITONEAL CAVITY INVOLVED IN</u>	<u>NO. of DEATHS</u>	<u>MORTALITY PER CENT.</u>
0- 5	4	2	1	25
6-10	35	25	3	8.6
11-15	51	28	4	7.8
16-20	57	24	3	5.3
21-30	82	31	2	2.4
31-40	39	18	1	2.6
41-50	18	10	1	5.5
51-60	10	5	1	10
61-	4	2	1	25

It will be observed that the percentage mortality reaches an acme in early childhood and in late adult life.

This, in the former, results from the cooperation of several factors, e.g.

The histological peculiarities.

The abundance of lymphoid tissue with absence of sub-mucous coat allows infection to pass more readily from the lymphoid follicles through the hiatus muscularis to the peritoneal coat.

Difficulties in diagnosis.

The early symptoms are not so clearly defined in the child and the attack is usually attributed to dietetic/

dietetic indiscretion.

The Violation of the law of rest to inflamed structures.

Practically every child is given aperients by its mother in the presence of abdominal pain. I am convinced that this is the explanation of the large number of perforated appendices found in children. Motley, in reviewing the results of his operation cases in children, was impressed with the very significant frequency of perforation following purgation. Of nineteen cases in which the appendix had perforated when the patients were admitted to hospital, sixteen gave a history of having been freely purged.

Adam says : "The mortality of appendicitis ever since it was first classified by the Registrar General as a cause of death has always been highest between the ages of 5 and 15. This corresponds to the period when the mother is most apt to dose her offspring with aperients and also that when the doctor finds the interpretation of symptoms extremely difficult."

Another factor, which I believe to be of importance in contributing to the high mortality, is the more vigorous peristalsis found in children.

Gunn and Whitelocke have investigated the movements of the appendix and they conclude that in their experience the most lively movements have occurred in appendices removed from young patients under 10 years of age.

In late adult life, the high mortality is due to failure/

failure to consider appendicitis as a possible cause of abdominal symptoms. Appendicitis is no respecter of person or of age and recent letters in the British Medical Journal have shewn that the condition is not at all infrequent even after 70 years of age.

The symptoms lack definition and Warren states that "the more elderly seem to have diminished sensibility so that they take little notice of pain in the abdomen until their condition has become serious. The pain of an incipient appendicitis is probably less severe than that of an intestinal colic from some dietary lapse, and is disregarded; it is surprising how slightly persons of a certain age show the severity of the trouble either subjectively or objectively."

Furthermore, toxins are less capable of being dealt with in that seventh stage where man is "sans teeth, sans eyes, sans taste, sans everything" (including resistance to infection.)

Sex.— The following table shows that the disease is more common in the male and has a higher mortality rate.

TABLE

	<u>CASES</u>	<u>DEATHS</u>	<u>MORTALITY PER CENT.</u>
M.	161	10	6.2
F.	139	7	5.04
TOTAL	300	17	5.7

Wilkie in investigating 1,469 cases of acute appendicular/

appendicular disease found that 57 per cent. were males and 43 per cent. females, while the death rate in the former was 9.6 per cent.; in the latter, 4.1 per cent. He considers there are two factors to explain this. First, the richer protein diet of the male, and second, the higher incidence in the former of strain and injury, "such as might determine the entrance of faecal material to an appendix with a stenosis or kink near its proximal end."

(c) Type of appendicitis.—

Of course it is obvious that acute appendicitis offers more immediate danger to life than does chronic appendicitis, but even in the acute form we find that there are certain types more dangerous than others. For example, in the catarrhal form, danger is at a minimum while in the perforative type a very serious condition has arisen which may endanger and eventually destroy the patient's life; it goes without saying that the less extensive the pathological process the less the danger to life.

Wilkie has brought to our notice a new classification. He regards acute appendicitis as being of two types, the acute inflammatory and the acute obstructive. At the Cleveland Meeting in 1926 he points out the difference in the prognosis of these two types thus :

"In acute appendicitis two conditions might be met with - acute obstruction and inflammation of the walls/

walls of the appendix. A small amount of faeces might be drawn into the lumen of the appendix, and form a concretion. The appendix might then become constricted retaining this concretion as a foreign body.

Under these conditions, gangrene may set in without causing fever, and perforation may occur within twelve hours. In the inflammatory group long before perforation occurs, a zone of reaction is set up and adhesions wall off the zone, while in the acute obstructive type adhesions have not had time to form, and the contents of the appendix rupture into the peritoneal cavity. The outlook is then very serious. There is also a group of cases coming in between these two, where there is nausea and possibly vomiting, pain in the region of the appendix and a rise of pulse and temperature. Twenty-four hours later, the patient has acute cramping pain in the abdomen which comes on spasmodically. Here inflammation has led to occlusion, and we have the picture of obstruction. Here, the consequences may not be so serious as the area is walled off by adhesions. The profession seems not to have grasped this fundamental difference in types of appendicitis; in Edinburgh, they have been making this differentiation since 1913. It was found that about five times as many deaths occurred in appendicular obstruction as in the primary inflammatory type of the disease, particularly among children.

In/

In dealing with the obstructive type, operation should be as prompt as possible; the incision should be a free one permitting removal of the appendix without rupture. The abdomen can then be closed without drainage even though the appendix be gangrenous."

Again, the virulence of the bacteriological agent will influence the prognosis. It is recognised that the worst types are met with in streptococcal infection although Williams' work also indicts the B. Welchii as producing gangrenous changes and toxic absorption. I am convinced that the position of the appendix is an important factor to be considered. My experience is that the pelvic appendix is concerned with the highest mortality. It is more difficult to diagnose and when diagnosed has often given place to pelvic peritonitis, which in turn readily produces 'ileus duplex'.

The Retro-caecal appendix usually gives a good prognosis, especially if it be retro-peritoneal; but a few may be associated with retro-peritoneal cellulitis, a condition difficult to control and of serious import.

The Mesenteric appendix is dangerous because it gives rise to early abdominal distension and may cause mesenteric thrombosis.

We have already seen that the clinical findings are not necessarily an accurate index of the severity of the pathological process and therefore do not form a/

a reliable prognostic guide.

(d) Treatment.—

This is by far the most important criterion. If operation is undertaken within the first twenty-four hours of onset and the appendix skilfully removed, then the prognosis immediate and ultimate is good. Early removal of the cause - if advisable - before the supervention of complications is a therapeutic ideal applicable to most diseases; and this is most desirable in acute appendicitis. Even Sherren - who is the exponent of conservative treatment in the later stages - advocates this. He says: "There is no exception to the rule that in acute appendicitis the appendix should always be removed when the disease is still confined to it; this is usually within the first twenty-four hours; occasionally we may be able to do it later. But if the patient has been seen medically, there is no need for even this delay; most could be removed within the first twelve hours and the earlier the better. Drainage is then unnecessary and work is resumed within a very short time."

To remove the appendix by early operation before the peritoneal cavity is involved is the surest way of reducing the mortality of this disease, which is, at present, still far too high. The details of treatment will be considered later.

(2)/

(2) Ultimate or late prognosis.

If the appendix has been removed before involvement of the peritoneal cavity, the ultimate prognosis in the large majority of cases is good. That this is not an invariable rule is shewn by the following history:-

T.E. male aged 28 years - had his appendix removed for acute appendicitis. Six months later he was readmitted to hospital complaining of pain over the splenic region radiating downwards and forwards towards the umbilicus; vomiting was persistent. At first it was thought he was suffering from renal colic but later a diagnosis of intestinal obstruction was made. This was confirmed by operation when a band was found strangulating a coil of small intestine. The band was divided, the gut was considered viable but unfortunately the patient died after operation.

If peritonitis has been present, the patient runs the risk of the following complications, some of which may prove fatal :

intestinal obstruction due to flakes of lymph binding down the intestine;

paralytic ileus which is often fatal unless a jejunostomy is performed;

faecal fistula which may cause death by dehydration;

sub-phrenic abscess;

femoral thrombosis;

ventral hernia especially where drainage has been prolonged.

In conclusion, each case must be considered on its merits and, while a cautious and guarded opinion should/

should be expressed in the presence of peritonitis, we should always try (in the words of Punch) to be neither a 'pooh-pooher' nor a 'wind-upper'.(Hutchison)

TREATMENT

This aspect of the subject bristles with difficulties. Conflicting opinions are met with everywhere. It is useful, therefore, to review briefly the evolution of the treatment suggested up to the present day. This is well summarised by Trotter thus: "At first appendicitis came into the province of the surgeon in the form of abscesses in the right iliac region which it was found could be satisfactorily treated by drainage. From these cases there gradually evolved the conception of the acutely inflamed appendix as the primary lesion and the regularization of operations to deal with it. A third phase was the recognition of incomplete spontaneous resolution, of the liability to recurrent attacks, and the appearance of the "interval" operation."

Sir Frederick Treves and Sir George Beatson in this country were among the early advocates of the 'interval' operation for all cases, but this attitude was challenged by the late Edmund Owen who, in his book, issued a plea for immediate operation.

This brings us to the present phase of treatment. A priori, the treatment of any disease requires the following considerations:-

- (1) Prophylaxis or prevention of the disease.
- (2) When the disease is established
 - (a) if advisable - removal of cause.
 - (b) if inadvisable - institution of physiological rest to the part.
- (3) Alleviation of symptoms.
- (4) Prevention - or treatment - of complications.

(1) Prophylaxis.

From the work of Short, Wilkie and Rosenow, the incidence of the disease would be considerably lessened by

- (i) eradication of primary foci of sepsis in teeth and tonsils.
- (ii) reducing the amount of protein diet (especially meat)
- (iii) increasing the amount of 'roughage' in the diet.
- (iv) prevention of constipation.

It has been advocated especially in Germany and America that appendicectomy should be performed as a routine in childhood, but this is not likely to become established in this country. But in the course of any abdominal operation - provided it does not endanger the patient's life - appendicectomy is to be advised, even for an apparently healthy appendix.

(3) The symptoms of any abdominal disorder should not be alleviated by morphia until a definite diagnosis has been made and acted upon. To give morphia in such conditions is to obliterate Nature's danger signals so that severe pathological processes may be at work but not revealed.

(2) and (4) - Since appendicitis is by far the most common cause of peritonitis, it brings the insignificant-looking appendix to the forefront in surgical importance. If appendicitis were properly treated, peritonitis would never be allowed to supervene. As patients die not from appendicitis as such but from peritonitis and its consequences, the mortality/

mortality from the disease, which is still ^{much} too high, would be reduced to a minimum if peritonitis were prevented.

The statistics in America and in this country show that the mortality from the disease is increasing. The Registrar General's returns show that in 1913 the deaths per million of the population from appendicitis were 69, while in 1923 they had increased to 74. Investigations are called for to consider the reason for this increase and to see how far it can be reduced.

There are two outstanding methods of treatment adopted at the onset of the disease. The first is the medical, or so-called Murphy-Ochsner treatment. This combines three of the most important therapeutic measures in this disease -

(a) The Fowler position introduced in 1900 by George R. Fowler. Here the patient is in a sitting up posture with the thigh and knees flexed. This posture was stated to be of use in confining (by force of gravity) infectious material to the lower portion of the abdomen whence absorption is much less rapid than in the diaphragmatic region. Although this theory has been challenged by Dandy and Rowntree, it is incontestable that the use of this posture in abdominal surgery has saved many lives.

(b) Murphy's operative technique and proctoclysis (1904). In the presence of spreading peritonitis, Murphy advocated speedy operation with limitation of intra abdominal manipulations which was crystallised in/

in his aphorism "Get in quick, get out quicker."

After operation he advised administration of large quantities of saline solution per rectum and he describes the technique as follows :-

"The saline should be administered through a fountain syringe to which is attached some $\frac{5}{8}$ inch rubber tubing fitted with a malleable metal rectal nozzle, having multiple openings. The nozzle should be fixed almost to right angles three inches from its tip.

"A straight tube must not be used, as the tip produces pressure on the posterior wall of the rectum when the patient is in the Fowler position.

"The tube is inserted into the rectum to the flexion angle, and secured in place by strips of adhesive plaster binding it to the side of the thigh, so that it cannot come out: the rubber tubing is passed under the bedding to the head or foot of the bed, to which the fountain is attached. It should be suspended from six to fourteen inches above the level of the buttocks, and raised or lowered to just balance hydrostatically the intra abdominal pressure, i.e. it must be just high enough to require from 40 - 60 minutes for one and a half pints to flow in, the usual quantity given every two hours.

"The flow must be controlled by gravity alone, and never by a forceps or constriction on the tube, so that when the patient endeavours to avoid flatus or strain, the fluid can rapidly flow back into the can; otherwise it/

it will be discharged into the bed.

"It is this ease of flow to and from the bowel that ensures against over distension and expulsion on to the linen.

"The temperature of the water in the fountain should be maintained at 100° Fahr. and it should be refilled every two hours with one and a half to two pints of solution. When the nurse complains that the solution is not being retained, it is certain it is not being properly given; even children tolerate proctoclysis surprisingly well."

(c) Ochsner (1904) considered the mortality in appendicitis to result from the extension of infection from the appendix to the peritoneum or from metastatic infection from the same source. The distribution or extension of the infection was accomplished by the peristaltic action of the small intestine, by operation after the infectious material had extended beyond the appendix and before it had become circumscribed. He says : "Peristalsis of the small intestine can be inhibited by prohibiting the use of every form of nourishment and cathartics by mouth and by employing gastric lavage in order to remove any substance of food or mucus from the stomach. The patient can be safely nourished during the necessary period of time by means of nutrient enemata. In cases where neither food nor cathartics are given from the beginning of the attack of acute appendicitis and gastric lavage is/

is employed, the mortality is reduced to an extremely low percentage. In patients who have received some form of food and cathartics during the early portion of the attack, and who are consequently suffering from a beginning diffuse peritonitis when they come under treatment, the mortality will still be less than 4 per cent. if peristalsis is inhibited by the use of gastric lavage and the absolute prohibition of all forms of nourishment and cathartics by mouth."

By combining the contributions of these three surgeons, viz., Sitting posture (Fowler), withholding of food and aperient (Ochsner), and the giving of saline solution per rectum (Murphy), it has been computed that 98 per cent. of cases of acute appendicitis can be tided over their first attack but the patient should have his appendix removed in the quiescent stage for 'once appendicitis, always appendicitis'.

The above method, then, is merely a preliminary to operative interference.

The second method is that of immediate operation. This was crystallised by the words of Osler in his classical text-book when he wrote: "There is no medicinal treatment for appendicitis."

The modern tendency is to operate as soon as the diagnosis is made because it is stated by many that no one knows what an appendix will do. For example, Kelly in his classical monograph states "Operation is safest because it can never be foreseen which cases will/

will go on to suppuration and which will not; moreover, fatal complications may arise at any moment, absolutely without warning."

Again, John B. Deaver writes: "There is no one who can say which case of acute appendicitis may progress to recovery and which go on to abscess formation or general peritonitis and death. This has been proved so often that we have no right to postpone operation in any case. It is true that a certain number of cases of acute appendicitis will recover spontaneously under proper non-operative treatment only to have subsequent attacks. It is equally true that some unoperated cases will die. Could we but differentiate the two classes clinically, our line of procedure would be easy to establish, but as has already been stated, such a differentiation is impossible, and the only proper and safe course, therefore, is to resort to prompt operation, when a case of appendicitis is seen early."

Again, Wilkie says that in certain cases acute appendicitis is a form of intestinal obstruction and, as such, should be operated upon immediately, thus: "I have no hesitation in including under the heading of acute intestinal obstruction a large group of acute affections of the appendix, and these are the most important because they are the most fatal if not recognised and operated on promptly. Until it is recognised and taught that a complete obstruction of the/

the lumen of the appendix is followed by changes which, depending on the nature of its content at the moment of obstruction, are either mild or hyperacute, and in the latter case are associated with early gangrene, we shall see no further reduction in the mortality of acute appendicitis, so called.

"I cannot refrain from referring once more to the fact -- which I have constantly taught for the past 12 years -- that the sudden locking up of faecal matter within a closed loop of intestine, be it small or large intestine or be it the appendix, is followed by rapid changes which culminate in gangrene and perforation of the loop. The changes are so rapid that an appendix may be gangrenous within six hours of being obstructed and this without any appreciable rise in the pulse or temperature of the patient."

The above views constrain us to think of the saying "A little fire is quickly trodden out,
Which being suffered, rivers cannot quench."

Presumably this is one of the occasions when, to use Moynihan's phrase "the physician is doomed to the practice of surgery". But is the above state of affairs true? Do we, or do we not know which case will go on to peritonitis and which will subside? While it is obviously true that early diagnosis followed by early removal of the appendix will rescue the patient from the dreaded complication of peritonitis, is this the only way in which this can be achieved?

The institution of the Murphy-Ochsner treatment is/

is followed by good results too and peritonitis prevented; and equally good results by both methods of treatment are reported by Scandinavian authorities. Are those methods necessarily self-contradictory or can they be explained on the basis of a common factor ?

I humbly submit that they can. The dominating factor of the whole situation is purgation.

Perforation of the appendix with subsequent peritonitis, in my experience, is invariably associated with the administration of an aperient; while, if the latter be omitted as in the Murphy-Ochsner treatment and physiological rest by withholding food and liquids given to the appendix, the attack will subside, although the organ will still be liable to subsequent attacks of inflammation.

This attitude towards the relationship of aperients and appendicitis was forced upon me by the various stages of my post-graduate experience which so far has consisted of three phases.

(1) Assistant in General practice with poor hospital accommodation, the nearest hospital more than twenty miles distant over very indifferent roads.

My chief, M.D., F.R.C.S.E., demonstrated that in these circumstances it was more advantageous to treat cases of acute appendicitis by the Murphy-Ochsner method, appendicectomy being performed in the quiescent period when the patient would be more able to undergo the ordeal of travelling. This treatment, so diametrically/

diametrically opposed to that advocated by my teachers, gave excellent results. I became a devotee to this method and all went well until I met one patient, a boy aged 12, whose condition was becoming worse under the treatment, with rising pulse rate and extending rigidity and tenderness. He was sent to Cardiff Infirmary where immediate operation revealed spreading peritonitis with a perforated gangrenous appendix, but fortunately he recovered.

My faith in the so-called medical treatment was now badly shaken, but on visiting the relatives, they apologised to me for disobeying my instructions. They had given the child doses of castor oil despite my warning that no purge should be given.

This case was the starting point of the conviction which I now strongly hold, that the whole treatment of appendicitis depends upon whether or not an aperient has been given. Subsequent experience has only served to strengthen this conviction.

The rule I adopted was this: If the case was seen early, the Murphy-Ochsner treatment was instituted with strict orders not to give an aperient; if an aperient had already been given, the patient was sent immediately to hospital. Very satisfactory results were obtained, the only disadvantage of the former being the greater loss of time to the patient, because of the subsequent appendicectomy.

(2) Next, a Cottage Hospital (25 beds) was opened/

opened in the district to which my Chief was appointed surgeon. We were now able to test my theory more severely.

If the patients were seen early and before an aperient had been taken, they were placed in the Fowler position, sips of water only given by mouth and no aperient, operation being deferred until the recognised operating day.

If an aperient had been taken, however, operation was performed immediately despite a normal temperature and pulse. We were amazed at the number of perforated gangrenous appendices found in these cases. It was only in such types that we 'knew no night or day' in the treatment of appendicitis.

The above régime also showed that the clinical findings cannot always be correlated with the pathological findings as revealed at operation.

(3) 1st Assistant Medical Officer to a General Hospital.

I now see many cases of acute appendicitis. If they have had an aperient I operate immediately but if the patient has been admitted during the night and has had no aperient, I employ the Murphy-Ochsner treatment together with morphia and operate the following morning. The results are eminently satisfactory and my experience can be summed up thus : I have yet to see a case of perforated appendix which has not been given an aperient.

These/

These views provoked considerable criticism from my colleagues but they too are now convinced that it is the administration of an aperient which produces perforation of an appendix.

I thought I had made a wonderful discovery because, unlike Deaver and Kelly, I could foretell what an appendix would do, provided I knew if a purgative had been given, but on looking up the literature I find that the authorities have long taught this view, e.g. :-

(1) Sir William Osler.- "The medical treatment of appendicitis can be expressed in three words - rest, opium, enemata. I would protest most earnestly against the indiscriminative use of saline purgatives, which have been advocated under a total misapprehension."

(2) Lord Moynihan.- "The symptoms one and all show a tendency to abatement if proper treatment is adopted, e.g., the patient is denied food of all kinds, fluid and solid, if aperients are strictly and sternly withheld. It seems to be a natural and instinctive desire of the mother, wife, or nurse, in such a condition, to administer forthwith a brisk cathartic. A few hours after the aperient is swallowed, frequently in the early hours of the morning, the patient is seized suddenly with a new and more intolerable agony. Vomiting occurs and diarrhoea may be repeated. The abdominal wall becomes rigid, tenderness spreads rapidly across the lower part of the belly, and at last/

last is everywhere present; the pulse rises steadily and all the signs and symptoms of acute peritonitis are ushered in without delay. When an operation is performed, a gangrenous and obstructed appendix is found.

"It is now about 14 years since I was first brought firmly to the conviction that in cases of appendicitis, it is the administration of an aperient that is responsible for the acute catastrophe of gangrene and perforation, which ends in acute peritonitis. I do not remember one single case that I have operated upon since, in which it was not perfectly clear that the same frequency of events - pain, aperient, perforation - had occurred, and I do not hesitate to say that in almost every instance of acute peritonitis due to perforation of an appendix, it is the treatment directed to the relief of the condition which is the cause of the serious and so often fatal catastrophe. The taking of a purgative medicine is something more than an impressive antecedent - it is in my judgement a definite cause."

(3) Deaver of Philadelphia.- "More dangerous than the surgeon's scalpel is the harmless looking cathartic pill or purgative draught, which is almost universally given in the early stages of the disease.

"The prevalence of this practice constitutes one of the greatest therapeutic follies of the day, and it is not too much to say that the majority of cases of/

of peritonitis resulting from appendicitis are instances of 'therapeutic peritonitis' due to medical maltreatment."

(4) Flint (Leeds).— "The patient, often a child and so unable to fend for itself, complains of bad stomach ache and is promptly given a dose of castor oil. There is no more certain way of spreading infection to the peritoneum than by stirring up the intestines with aperients at the beginning of a local inflammation such as appendicitis; when it is seen that the pain is worse the doctor is summoned, and by this time probably the appendix has perforated, and of course the doctor or surgeon receives the blame if the result of an operation is unsatisfactory. I recently advocated a campaign of public education properly controlled in certain medical matters. I feel convinced that without it progress will be terribly slow. Even if only one statement were thoroughly driven home, namely, that it is dangerous to give an aperient for acute abdominal pain without the sanction of a doctor, I feel sure we should see an immense and immediate improvement in the outlook for the acute abdomen".

(5) McNeill Love (London), in his Hunterian Lecture quotes Adam's statement, viz. "The mother may be an unconscious murderer of her own child, and a dose of castor oil may be as poisonous as a dose of Hemlock".

I have also referred to the text-book on Medicine by Fagge which was considered the authoritative work some fifty years ago, and it is interesting to quote his description.

"The course of 'typhlitis' depends, I believe, very largely upon the treatment which is adopted. I have indeed a strong conviction that no case in which the disease can be diagnosed ever terminates fatally if judiciously managed. The essential points are that the patient should be kept perfectly at rest, that his diet should be strictly limited to slops, that he should not be allowed to take a single dose of aperient medicine and that opium should be given freely. And when the attack subsides the greatest care must be taken to prevent a relapse. Even then, the action of the bowels should be solicited by enemata only and never by medicines taken into the stomach."

In my opinion this is an excellent outline of the treatment to be adopted.

Wilkie distinguishes two types of acute appendicitis; the acute inflammatory, the greater number of which will recover without operation, and the acute obstructive in which gangrene and perforation readily occur and for which immediate operation is necessary. I have recognised this difference in types as described by Wilkie, but only at operation; I have never been able to satisfy myself that I can distinguish them by clinical examination.

I submit with the greatest possible respect that the two types can be better differentiated from the history of purgation. I believe that the obstructive type results from the action of aperients. If obstruction of the lumen be present either from a faecolith, stricture or oedema of mucous membrane, the purge causes marked spasmodic contraction of the appendix and perforation results. (This is analogous to rupture of the uterus after prescribing pituitrin in cases of obstructed labour.)

If the case be originally of the inflammatory type, the aperient forces more faecal content into the lumen of the appendix and the inflammatory now gives way to the obstructive variety with subsequent perforation. This would also explain the large amount of faecal material which Wilkie associates with the obstructive forms.

Early operation is recommended by surgeons because peritonitis is seldom found before the first twelve hours. The reason for this, I submit, is that aperients seldom act before the elapse of this time.

The General Practitioner will not take the responsibility of treating appendicitis; he cannot differentiate between those which will subside and those which will go on to perforation and he rightly sends the patient straightway to hospital. The surgeon associates the gangrenous and perforated forms with delay in sending the patient to hospital, and he therefore/

therefore always advises early operation. Thus, I believe, few realise that it is the time necessary for the action of the aperients which determines whether the case of acute appendicitis will progress to perforation and spreading peritonitis.

Again, as Grey Turner of Newcastle has pointed out, the march of pathological processes in appendicitis is not to be measured by the clock. To this I agree, with this reservation - that if an aperient has been given then the longer the interval between the aperient and the operation, the more extensive the peritoneal involvement.

I have drawn up in the Analysis, a table which bears this out; the longer the interval after the aperient, the more diffuse the peritonitis and the higher the mortality.

There are, in my opinion, two ways of eliminating the development of peritonitis in cases of appendicitis -

- (1) Omission of purgation.
- (2) Early operation, i.e. before aperient, if taken, could act.

This explains why the Murphy-Ochsner treatment or early operation give equally good results.

By early operation and by omission of purgation, we are making doubly sure that peritonitis will not supervene. Furthermore, early operation removes the only disadvantage of the Murphy-Ochsner treatment, viz., the necessity for subsequent appendicectomy.

The points which I wish to make clear are :-

1. In districts with good hospital accommodation, early operation and omission of purgatives is the ideal and will reduce the mortality to a minimum.
2. In districts far removed from hospital, or during a sea voyage, it is possible to obtain equally good results by the Murphy-Ochsner treatment (aperients being prohibited) with subsequent 'interval' appendicectomy. This latter treatment could also be applied where operation, for the time being, was contra indicated, e.g., business reasons, or during any other acute illness.

There still remains to describe very briefly the operative technique employed once operation has been decided together with a resumé of the measures usually adopted to save life when peritonitis has occurred.

Pre-operative.

There seems to be a tendency to restrict the use of the Fowler position to post-operative treatment alone but this is surely wrong. The assumption of this posture before operation is as likely to yield the same beneficial effects as after operation. As soon as the diagnosis of appendicitis is made, the Fowler position should be used forthwith.

It would appear, a priori, that, if many cases are streptococcal in origin, the use of anti-streptococcal serum should be beneficial but it seems to have little effect.

Wilkie advocates vaccines of B. coli communis and injections of nucleic acid which produces leucocytosis.

Operation/

Operation.

The diagram in the accompanying atlas, which is taken from Kelly's classical work, shows how many and varied are the incisions used in operations upon the appendix. Mr. Miles (Edinburgh) in his clinical lectures used ironically to tell us that a surgeon had not won his spurs unless he had described a new method of approach to the appendix !

There are three important incisions.-

First, the McBurney or gridiron incision, which should be used where the disease is localised to the right iliac fossa.

I nearly always use this incision, which is practically never followed by incisional hernia.

Second, Battle's incision through the rectus sheath. This is stated to be of use in the female to allow of exploration of the pelvis, but it has the following disadvantages:-

It is liable to be followed by ventral hernia due to division of nerves.

Suppuration of the rectus sheath may follow if peritonitis is present.

Secondary haemorrhage may occur from pressure of drainage tube on the inferior epigastric vessels.

I seldom use the above incision, preferring, in cases of pelvic peritonitis, to use the

Third, or paramedian incision.

This allows of extension of the incision without damage to the anterior abdominal wall, and with complete inspection of the pelvic viscera.

The other details are as described in the textbooks, /

text-books, except that I prefer the N-shaped suture for burying the appendix stump.

Post-operative care.

The patient is placed in the Fowler position as soon as he has recovered from anaesthesia, sips of water are given by mouth and no aperient is given.

I lost one patient through the sister giving an aperient on the second day after operation and causing extrusion of the appendix stump and spreading peritonitis. Quarry Wood of Edinburgh refers to a similar case.

The bowels are to be solicited by enemata (compare Fagge) according to the dictum: Look after the large intestine and the small intestine will look after itself.

The patient is usually allowed up on the tenth to the fourteenth day and discharged a few days later.

BRIEF RESUMÉ OF TREATMENT IF PERITONITIS IS PRESENT AT OPERATION.

If appendix abscess be present, the abscess may be merely drained, appendicectomy being performed later. This is favoured by Adams who states that "all that is required is relief of intra-peritoneal tension."

That this is not always without danger is shewn by the fact that two of my patients treated on the above lines developed heart block (pulse rates being

36 and 48.), while I have already referred to the patient who developed septic arthritis of the spine.

These cases show that toxic absorption may still continue and produce evil general effects.

I only leave the appendix behind in these cases if it is buried in adhesions and am inclined to agree with Rendal Short when he says :

"An experienced surgeon will very, very seldom leave the appendix; the occasional operator, if he is wise will do so frequently. If the appendix is left, about 17 per cent. of the patients will have further trouble."

If spreading peritonitis be present, we have again two lines of treatment advocated.

1. Immediate operation.
2. Delayed operation.

This latter method has been advocated by Ochsner, Sherren, Berry, and more recently supported by McNeill Love and Hamilton Bailey. Ochsner has already been quoted as stating that the mortality in cases of commencing diffuse peritonitis will be less than 4 per cent. if peristalsis is inhibited by the use of gastric lavage and the absolute prohibition of all forms of nourishment and cathartics my mouth.

Sherren in 1905 stated that better results were obtained after the third to the fifth day of the disease when general peritonitis was present, by delayed operation. The patient is to be placed in the/

the Fowler position and nothing but water given by mouth while fomentations are applied to the abdomen to relieve pain. The mortality rate in such cases was stated to be less than if operation had been performed.

Sir James Berry in his presidential address to the Royal Society of Medicine says : "An operation which I think is performed too frequently is that for appendicitis. It is of course quite true that the great majority of operations upon the quiescent appendix are easy of performance and almost devoid of danger. But there is a minority in which the operation may be a very severe and difficult one and the difficulties both in diagnosis and in performance are not easily foretold by the operator. Where I venture to think most harm is done is when the case is not seen in the very early stage of the disease and especially when the stage of abdominal distension has already been reached. Most of such cases do not die if not operated upon and the delay of a few days often puts the patient in a much more favourable condition for operation if it has to be undertaken. In an address which I gave fifteen years ago after much study and experience of the subject, I expressed the view that if we were all to go back to the old treatment of appendicitis that was in vogue 25 (now 40) years ago, there would be a lesser mortality from appendicitis than there is at present. Fifteen years further/

further experience of the subject has confirmed me in the opinion I then expressed."

McNeill Love, from statistics of the London Hospital argues that Sherren's method of delayed operation is better in cases of spreading peritonitis and Hamilton Bailey of Birmingham has recently written supporting this treatment which he refers to as the Ochsner-Sherren treatment.

The value of the above treatment is still under review and has produced considerable discussion and criticism, especially from the late Joseph Adams who issues a warning against reverting to the old state of affairs "for what the hospitals do today the general practitioner will do tomorrow."

The majority are more inclined to operation and I think it is more correct to say of a spreading peritonitis than of an appendix that 'no one knows what it will do'. If operation be undertaken in these cases Murphy's counsel of 'get in quick, get out quicker' should be remembered.

PREVENTION OF EVIL EFFECTS OF PERITONITIS.

We have already seen that there are two main dangers -

- (1) the absorption of toxins.
- (2) the power of the peritoneum to form adhesions which may cause intestinal obstruction.

Very briefly we will consider the methods described to deal with those dangers.

(1)/

(1) Toxic absorption may be lessened by drainage. The indications for and against drainage have been keenly debated. Carslaw, Wilkie and St. Leger Brockman have discussed these indications. Wilkie states that an immediate examination of the peritoneal fluid forms the guide when to drain. He states that drainage is indicated when the large mononuclear cells are diminished and fail to stain, and also by the absence of phagocytosis. Brockman in a recent Hunterian Lecture suggests that drainage is too often employed. He reports 352 cases of gangrenous appendicitis in which the mortality was 8.5 per cent. when a drainage tube was used, and 192 undrained cases with a mortality of 7.2 per cent.

At present, I still adhere to the dictum "when in doubt, drain".

The question as to whether irrigation of the peritoneal cavity should be employed to wash out toxins is also debated. Mackechnie states that "non-irrigation has the advantage of not disturbing adhesions, of not opening up closed avenues of absorption and of not traumatizing tissues, and of not washing away the antibodies developed to take care of the existing infection. Irrigation has the advantage of removing a large quantity of septic material, of producing a large flow of blood to the abdomen and with it an increase of antibodies; it dilutes the toxins being absorbed and it always decreases the dreaded/

dreaded ileus of these cases."

Rulison believes better results are followed by the injection of Dakin's solution abdominally while Saliba favours the injection of ether into the peritoneal cavity.

Costain's lymphaticostomy is now discontinued but large fomentations over the liver increase the powers of that organ to neutralise toxins carried to it by the portal vein, and morphia by lessening the intestinal peristalsis is also stated to reduce toxic absorption.

Williams, who believes that the toxins are absorbed from the lumen of the bowel, advised the use of anti-gasgangrene serum and this has given good effects in my own cases.

Murphy's proctoclysis also produces a dilution of circulating toxins.

(2) Prevention of adhesions has been advocated by the use of sterile oil or vaseline (Wilkie) or by the use of citrate (Pope; Walker and Ferguson.)

The two dreaded complications are paralytic ileus and intestinal obstruction. Sampson Handley has described the condition of 'ileus duplex' in cases of pelvic peritonitis. He disapproves of jejunostomy (Victor Bonney) for these cases and advocates a short circuiting operation combined with a temporary caecostomy; in the pelvic stage an ileo caecostomy with caecostomy; in the hypogastric stage a jejuno-colostomy/

jejuno-colostomy with caecostomy.

Wilkie describes a unique case in which a double enterostomy was performed, the upper tube leading into the jejunum and the lower into the ileum. To prevent physiological starvation he fed the patient through the upper tube and later connected the two tubes by an extra-abdominal glass tube. The patient recovered and it is interesting to note that according to Wilkie's description, the patient had been previously given an aperient.

Large doses of anti-gas gangrene serum are useful in preventing the onset of paralytic ileus and I prescribe it as a routine in all cases of peritonitis.

The second dreaded complication is intestinal obstruction. This may be due to obstruction by a band or flakes of lymph. St. Leger Brockman in the Arris and Gale Lecture (1927) reports favourably on the use of enemata of bile to diminish persistent vomiting in these cases.

It is outwith the scope of this Thesis to discuss other complications such as faecal fistula, portal or femoral phlebitis or ventral hernia. I have tried to indicate how peritonitis can be prevented by early operation and the omission of purgation.

If these two rules are carried out, it will go far to reduce the high mortality of acute appendicitis and will help to bring about the happy state of affairs/

affairs visualised by Dieulafoy when he said :

"There should be no death in appendicitis."

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S U M M A R Y.

The compulsory modification of the treatment of acute appendicitis to meet the requirements of a large industrial practice, far removed from hospital accommodation, enforced me to take a keen interest in the problem of this disease. Contrary to the teaching instilled in me during my undergraduate days, I saw that many acute cases could be tided over without operation. The fact that every case did not respond to non-operative régime raised the question : Why do some resolve while others proceed to peritonitis?

Since then, I have formulated a theory which has borne the test of time and which seems to solve many of the problems met with in the disease. This truth was forced upon me by special circumstances (1) in General Practice with no hospital accommodation -- medical treatment was largely adopted; (2) in General Practice with an attached hospital -- early diagnosis and early operation, if indicated; (3) in a General Hospital -- receiving cases where the disease was well established and many with general peritonitis.

This truth explains, first, under what circumstances cases treated medically will recover; second, why immediate operation gives good results; and third, the factor producing peritonitis.

The Thesis presents the following :-

(1) The chief factors influencing the position of/

of the appendix, embryological and topographical; histological structure of the appendix; the abundance of lymphoid tissue; precarious blood supply and the communication via lymph supply with stomach, duodenum and gall-bladder.

(2) The functional importance of the appendix is considered from phylogenetic and structural grounds as well as from the effects of its removal. The conclusion arrived at is that we can believe that the appendix has a function yet it is essentially vestigial in character and that its potentialities for harm are greater than its capability for good.

(3) Aetiology is considered, appendicitis being due to a streptococcus borne via the blood stream from the teeth or tonsils. The appendix being a locus minoris resistentiae forms a nidus for those circulatory micro-organisms. Protein diet is an important predisposing factor.

(4) The various pathological lesions are described from my own specimens and sections. Spread of the disease occurs

- (a) by contiguity of tissue to the peritoneum.
- (b) by blood stream to the liver.
- (c) by lymphatic stream to the stomach, duodenum and gall-bladder.

The physiology of the peritoneum is considered and its powers of absorption and of forming adhesions lead in turn to toxic absorption and to intestinal obstruction if the infection spreads from the appendix to the peritoneal cavity.

The means of limiting toxic absorption are discussed.

(5) The symptoms of the condition are inflammatory or obstructive in origin. The value and significance of each symptom is assessed and fallacies in their interpretation pointed out.

The three outstanding symptoms are Pain which is of two types (i) initial or splanchnic (ii) secondary or due to involvement of the parietal peritoneum; Tenderness; and Rigidity.

It is stressed that pain does not usually begin in the right iliac fossa.

The severity of the symptoms can not be correlated with the severity of the pathological process.

(6) Actual and differential diagnosis are considered and Murphy's triad - epigastric pain, vomiting, and pain in the right iliac fossa - emphasised.

A warning against placing reliance on increase of temperature or a rapid pulse as positive evidence of acute appendicitis is given.

The method of examination, importance of pelvic or rectal examination, are alluded to and the various conditions from which appendicitis is to be differentiated are tabulated, special reference being made in the case of children and women.

(7) The unsatisfactory condition of the diagnosis and treatment of chronic appendicitis is stressed and a plea made for a more thorough clinical examination/

examination combined with an operative incision which will allow examination of other abdominal viscera.

(8) The various factors concerned in prognosis are discussed, reduction of mortality being dependent upon prevention of peritonitis.

As regards treatment, it is suggested that the whole key to the situation lies in the administration or withholding of an aperient.

The Murphy-Ochsner treatment is feasible because purgation is prohibited. Surgeons advise early operation before purgation has had time to act, i.e., before peritonitis has set in.

These two methods of treatment have one common factor - a purgative.

My experience is summed up in the statement: I have yet to see a case of perforated appendicitis which has not had an aperient.

Various authorities are quoted in support of the view that "the purge is more dangerous than the scalpel".

By early operation and by omission of a purgative, we are making doubly sure that peritonitis will not supervene; also early operation prevents the possibility of subsequent attacks.

A short analysis of my series of 600 cases is given.

ANALYSIS of CASESA. 300 Acute Cases Treated By Operation.

TABLE I.
(Shewing Age incidence and Mortality.)

<u>AGE</u>	<u>NO. of CASES</u>	<u>PERITONEAL CAVITY INVOLVED IN</u>	<u>NO. of DEATHS</u>	<u>MORTALITY PER CENT.</u>
0 - 5	4	2	1	25
6 -10	35	25	3	8.6
11 -15	51	28	4	7.8
16 -20	57	24	3	5.3
21 -30	82	31	2	2.4
31 -40	39	18	1	2.6
41 -50	18	10	1	5.5
51 -60	10	5	1	10
61 -	4	2	1	25

TABLE II.
(Shewing Sex incidence and Mortality).

	<u>CASES</u>	<u>DEATHS</u>	<u>MORTALITY PER CENT.</u>
M.	161	10	6.2
F.	139	7	5.04
TOTAL	300	17	5.7

TABLE III./

TABLE III.

(Shewing Relation of Time Interval between
aperient and operation to Mortality
(where peritoneal cavity is involved.)

<u>INTERVAL</u> <u>BETWEEN APERIENT</u> <u>and</u> <u>OPERATION.</u>	<u>RECOVERIES</u>	<u>DEATHS</u>	<u>MORTALITY</u> <u>PER CENT.</u>
1 - 24 hours	6	0	0
25 - 48 "	28	2	7.8
49 - 72 "	41	5	12.2
72 -	54	9	16.6
TOTAL	129	16	12.3

TABLE IV.

(Cases of Involvement of peritoneal cavity)

	<u>NO.</u> <u>of</u> <u>CASES.</u>	<u>RECOVERIES</u>	<u>DEATHS</u>	<u>MORTALITY</u> <u>PER CENT.</u>
Appendix Abscess	39	36	3	7.4
Spreading Peritonitis	106	93	13	12.3
<u>TOTAL CASES</u> <u>of</u> <u>PERITONITIS</u>	145	129	16	11

TABLE V.

(Analysis of Deaths.)

Acute Appendicitis	=	1
Appendix abscess	=	3
General peritonitis	=	13
<u>TOTAL</u>	=	<u>17</u>

Mortality per cent in 300 cases = 5.7

B. Cases of Acute Appendicitis
treated by Murphy-Ochsner
method 49 : No deaths.

Cases of Chronic Appendicitis = 83

Cases of appendicectomy as
part of other operations. 168

Prophylactic	Diseased Appendices
59	109

Total Number of Cases = 600.
